

STUDIES ON THE PATHOLOGY AND TREATMENT OF
EQUINE NAVICULAR DISEASE

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February 1984.



I declare that the contents of this thesis are my own work and have not been presented to any University other than the University of Edinburgh.

February 1984.

To my Family and Friends

"Shall I tell you what true knowledge is?
When you know, to know what you know,
And when you do not know, to know that
You do not know - that is true knowledge."

Confucious

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ACKNOWLEDGEMENTS

I wish to thank Professor J R Campbell for his help and support, and for the facilities made available to me in his department. I am indebted to Mr J A Fraser for his invaluable advice, encouragement and undying patience throughout this project.

I would like to thank Mr W Wilson for his skilled assistance and helpful advice in the handling of the horses in this study, and Mr A Riley for his assistance with the treatments.

I am most grateful to Mrs G McHarrie, Miss E Grieve and Mrs M Haworth for their skilled technical assistance with the radiography and angiography, and to Mrs C Page for her help in cutting undecalcified bone sections and histological sections.

I wish to thank Mr A C Rowland for his invaluable advice with the pathological studies, Mr D Ritchie and Mr M Slater for their assistance in obtaining post-mortem specimens, and to the Department of Veterinary Pathology for the use of their facilities.

My thanks to Mr R Munro and Miss F Manson for the photography, and to Mrs H London for her cheerful assistance with the references.

I am grateful to the grooms of the R.(D).S.V.S. Veterinary Hospital for their help in the care and handling of the horses in this study.

For the unenviable task of typing the manuscript I wish to thank Mrs S Purves and Mrs S Hayton, and for producing the numerous tables and appendices Mrs J MacDonald and Miss P McManus.

Finally I am indebted to the Horse Race Betting Levy Board for their generous financial support which made this study possible, and to the Royal College of Veterinary Surgeons, and the trustees of the R.(D).S.V.S., Centenary Postgraduate Fellowship, for their financial

assistance in the last year of this work.

To all of these people and to many more unmentioned I express my sincere thanks.

SUMMARY

This present study arose following the hypothesis that vascular occlusion and progressive ischaemia were involved in the pathogenesis of navicular disease. The study was designed so that an evaluation of the radiographic and pathological changes in the navicular bones and surrounding structures could be carried out in horses in which a clinical diagnosis of navicular disease had been made and these compared with the results of similar examinations in control horses with no evidence of lameness. Using clinical criteria combined with an objective method of assessment based on radiographic findings, the effects of different treatments of navicular disease were examined with particular emphasis on oral warfarin therapy.

Of the 265 horses examined clinically, 90 constituted a control population, 151 had a lameness which was diagnosed as navicular disease and the remaining 24 horses, although they displayed a fore foot lameness similar to navicular disease, it fulfilled less than half the required clinical criteria and they were thus designated "not yet diagnosed" NYD.

No one clinical feature was diagnostic of navicular disease in itself but it was concluded that the clinical diagnosis should be based on the presence of a number of clinical signs, and the response to a number of diagnostic aids.

It is considered from this study that the particular type of work performed by a horse does not in itself predispose the animal to navicular disease but that irregular work, and sudden or prolonged periods of rest in an otherwise hard worked horse are predisposing factors.

From radiographic examinations of the navicular bones of the

horses in this study, no particular radiographic feature was considered to be diagnostic of navicular disease. The significant radiographic changes in the navicular bone in navicular disease were, an increase in number and change in shape and distribution of the distal nutrient foramina, the presence of nutrient foramina in the proximal border and radiolucent areas in the body of the bone. Some of these radiographic changes were found in the navicular bones of the control horses.

The distal nutrient foramina were identified and classified according to their morphology and position and 7 basic types were found. Distal nutrient foramina, shaped other than conical were considered as abnormal.

Examination of serial radiographs at intervals showed that the distal nutrient foramina could increase in number and show changes in their shape which were part of a progressive series of changes.

An objective method of assessing radiographs of the navicular bone was developed which consisted of a scoring system based on the number shape and anatomical position of the distal nutrient foramina. Significantly higher scores per navicular bone were found in the navicular disease group than in the control group, although there was a degree of overlap between the groups. It is considered that this navicular scoring system provides a useful method of evaluating radiographs of the navicular bone, but should not be used as the sole criterion in the diagnosis of navicular disease.

Pathological examination, which included gross pathology, radiography, microangiography, fluorescent microscopy following the administration of intra-vital fluorochrome bone labels to a number of horses, and light microscopy, showed that pathological changes

were present in all of the navicular bones and deep flexor tendons in the navicular disease cases, and also in a small number of control cases. These pathological changes were present to different degrees and could be fitted into a pattern of increasing severity. The main pathological changes found in the navicular bone were, degeneration of the fibrocartilage, disruption and loss of the subchondral bone plate of the fibrocartilage surface, an overall increase in the vascularisation, remodelling of the trabeculae in the medulla, and in the deep flexor tendon there was disruption of tendon fibres. A significant positive correlation was found between the degree of gross pathological change present in the navicular bones and deep flexor tendons and the navicular score.

No evidence of occlusive vascular disease or of bone necrosis was found in any of the navicular bones examined in this study. These results do not therefore support the hypothesis that occlusive vascular disease and progressive ischaemia are involved in the pathogenesis of navicular disease. However, the overall increase in vascularisation and active bone remodelling, in the absence of obvious vascular occlusion and bone necrosis suggests that hyperaemia rather than ischaemia may be involved in the pathogenesis of this condition. Although the cause of the hyperaemia has not been established, there is evidence that active and/or passive hyperaemia could be involved.

The treatments used in this study were, rest, oral warfarin therapy, corrective shoeing, phenylbutazone, neurectomy and isoxsuprine hydrochloride. The results of the treatments were assessed clinically and radiographically using the navicular scoring system. No significant difference was found between the results of the different

treatments at 3-4, and 6-8 months from the start of treatments, but significantly better results were found with warfarin therapy, 1 year from the start of treatments.

It is considered from the results of this study that the positive effects of warfarin and isoxsuprine hydrochloride in the treatment of navicular disease may not be related to the anticoagulant or vasodilator effects but may be associated with their ability to reduce blood viscosity and thus improve blood flow.

ABBREVIATIONS

R.(D.)S.V.S.	Royal (Dick) School of Veterinary Studies
NYD	Not yet diagnosed
kV	Kilovolts
mAS	Milliamps per second
N.V.L.	No visible lesion
OSPT	One stage prothrombin time
AST	Aminoaspartate transferase
γGT	γ-Glutamyl transferase

CHAPTER 1
GENERAL INTRODUCTION

He who does not know the aim cannot know the way.

V F F BLAKE.

THE AIM

The aims of the work described in this thesis are:

- (i) To investigate the clinical and radiological manifestations considered to be due to navicular disease in the horse.
- (ii) To evaluate the bone and vascular changes in the navicular bones of normal horses and those with evidence of navicular disease, and to relate these changes to the clinical and radiological findings.
- (iii) To investigate the hypothesis that navicular disease is the result of vascular occlusion and that treatment with oral warfarin therapy is effective by relieving ischaemia.
- (iv) To examine the clinical, radiological, and pathological changes following oral warfarin therapy and other treatment regimes.

THE DISEASE

Navicular disease is one of the commonest causes of chronic foreleg lameness in the riding horse. It was first recorded as a disease entity 230 years ago by Jeremiah Bridges (1752), in his essay "No Foot No Horse". The disease affects the navicular or distal sesamoid bones of Equidae, and would appear to be species specific.

ANATOMY

The navicular or distal sesamoid bone, is a shuttle shaped bone which lies entirely with the hoof on the posterior aspect of the pedal joint (FIG 1.1). It has two surfaces. The articular surface faces upwards and forwards. It is covered with articular cartilage and articulates with the distal posterior surface of the second phalanx. The flexor or tendinous surface faces downwards and backwards. It is covered with fibrocartilage and is the surface over which the deep flexor tendon runs. Both surfaces have a central prominence or ridge running at right angles to the long axis of the bone. It is considered that the articular surface of the navicular bone is not affected in navicular disease, whereas the tendinous surface is affected (Hickman, 1964).

The navicular bone has proximal and distal borders to which are attached the suspensory navicular ligament, and the distal navicular ligament respectively. Running through these ligaments are the nutrient arteries that supply the navicular bone.

Three quarters of the navicular bones surface is surrounded by fluid filled cavities, (Colles, 1982), the pedal joint on one side and the navicular bursa on the other (FIG 1.2). Of the so called "navicular bursa" Hickman (1964) wrote;

It is generally supposed that a bursa is interposed between the tendinous surface of the bone and the deep flexor tendon. This is not so; the area abounded by the joint capsule of the corona-pedal joint, the tendinous surface of the navicular bone, the interosseous ligament and the deep flexor tendon is a potential cavity lubricated with synovia, and as it is not lined by any separate and distinct endothelial membrane it therefore constitutes a joint.

FUNCTION OF THE NAVICULAR BONE

Two different functions have been attributed to the navicular bone. Fitzwygram (1894) and Rooney (1967) maintained that the navicular bone was a true sesamoid bone, its purpose being to provide a constant angle of insertion for the deep flexor tendon on the third phalanx. Others such as Reeks (1925), Hickman (1964) and Adams (1974) considered that the navicular bone was part of the shock absorbing mechanism of the foot. Colles (1982) showed that the navicular bone moved considerably during a normal step and suggested that;

The increased flexibility of the joint afforded by the moving navicular bone will allow it to absorb concussion whilst reducing the trauma that would be present in the posterior part of a rigidly boned joint.

CLINICAL FEATURES OF NAVICULAR DISEASE

Navicular disease can be defined clinically as a chronic, progressive, unilateral, but more often bilateral, condition of the front feet. It most commonly affects adult horses between 6 and 12 years old, although it is by no means confined to this age group. The disease results in a characteristic lameness, which is intermittent in its early stages and persistent in its later stages. As a result of a painful lesion in the posterior part of the foot, there is reduced weight bearing on the heels. The clinical signs include shortening of the stride, tripping, pointing of the affected limb or limbs, and an alteration in hoof conformation. For diagnostic

purposes, the lameness can be relieved or improved in most cases following the infiltration of local anaesthetic around the medial and lateral palmar digital nerves which supply the posterior part of the foot. Radiography plays an important part in the diagnosis of the condition, affected navicular bones showing a variety of specific changes.

AETIOLOGY AND PATHOGENESIS OF NAVICULAR DISEASE

Opinions vary considerably as to both the aetiology and pathogenesis of the disease. Some workers such as Oxspring (1935) and Rooney (1974) considered that the first pathological change was erosion of the fibrocartilage on the tendinous surface of the navicular bone, followed by changes in the bone itself. Erosion of the overlying deep flexor tendon was thought to occur at the same time and was considered to be secondary to the cartilage damage. These lesions were reported to be mechanical/traumatic in their origin.

As early as 1885, Professor Walley in a paper to the Midland Counties Veterinary Medical Association said of the origins of navicular disease; "I have long held the opinion that the anatomical position of the navicular bone is such as to predispose it to grave interference with its intra-osseous circulation". He was of the opinion that this interference with circulation arose as a result of working horses at a fast pace, and then pulling them up sharply;

This system of treatment is seen in perfection in horses belonging to butchers, in trotting and in cab horses, and I may also say in those belonging to veterinary surgeons and to men whose time is occupied by driving their horses to the top of their pace for the sake of effect, and suddenly pulling up every three or four miles for the purpose of refreshing the inner man.

Several workers since then (Jones, 1938; Hickman, 1964;

Nemeth, 1972; Colles and Hickman, 1977 and others) have suggested that changes in the blood supply to the navicular bone were involved in the pathogenesis of the condition. In recent years there has been considerable evidence (Nemeth, 1972; Colles and Hickman, 1977; Fricker, Riek and Hugelshofer, 1982) that vascular occlusion is closely associated with the development of navicular disease and possibly sesamoiditis in the horse. Investigations by Colles and Hickman (1977) into the blood supply of the navicular bone in normal horses and those with navicular disease, have shown that evidence of occlusive vascular disease, in the form of thrombosis and arteriosclerosis, was consistently present in a number of arteries within the navicular bones of the diseased cases. The same vascular changes were also encountered in a small number of the normal navicular bones examined (Colles, 1979).

These findings led to the hypothesis that, as a result of the obstructive vascular changes and reduced blood flow, areas of partial ischaemia could develop within the navicular bone. A secondary or compensatory blood supply then develops from the already present periosteal blood vessels along the distal border of the bone, and this in turn may become involved in thrombosis. If this vascular occlusion proceeds faster than the revascularisation, then progressive ischaemia and bone necrosis will develop (Colles, 1982). These necrotic areas can involve the subchondral bone of the tendinous surface with its subsequent collapse and cavity formation.

Since it is known that ischaemia of soft tissue is painful, (Lewis, 1942) and that pain results from ischaemia of the scaphoid bone in man, it was suggested by Colles and Hickman in 1977 that ischaemia of the navicular bone also resulted in pain and so lameness. In

contrast to this, the avascular necrotic bone lesions occurring in Caisson disease of man do not cause pain unless these involve the subchondral bone of the articular surface as in the femoral head (Cox, 1974). Caisson disease of bone is a condition of compressed air workers and divers, in which it is thought that gas emboli occlude the arterioles to specific areas of bone.

Considerable difference in opinion still exists regarding the aetiology of the navicular disease lesions, between the advocates of the mechanical/traumatic theory, (Østblum, Lund and Melsen, 1982) and those of the ischaemic necrosis theory (Fricker, Riek and Hugelshofer 1982).

RADIOGRAPHIC CHANGES IN NAVICULAR DISEASE

Opinions vary also, as to the significance of the various radiographic changes in the navicular bone associated with navicular disease, and as to the relationship between these changes, the degree of clinical involvement and the extent of the pathological lesions found.

Radiography has been used routinely as a diagnostic aid in navicular disease for the past 50 years. No single radiographic lesion is in itself diagnostic of the condition. Some workers such as Carlson (1977) and Adams (1974) maintained that in many cases of navicular disease no radiographic changes could be found while others like Oxspring (1935), Olsson (1954) and Colles (1982) considered that in all cases of navicular disease some radiographic changes could be demonstrated. Wintzer (1964) believed that the early radiographic changes could be demonstrated before the onset of clinical signs.

The radiographic changes generally agreed as indicative of

navicular disease are:

- (i) An alteration from normal in the shape of the nutrient foramina in the distal border of the bone.
- (ii) An increase in the number of nutrient foramina in the distal border of the bone.
- (iii) Areas of osteoporosis within the navicular bone substance.
- (iv) New bone growth on the medial and lateral extremities of the proximal border of the bone.

It is thought however that these changes can also be encountered in clinically sound horses.

TREATMENT OF NAVICULAR DISEASE

The treatments for navicular disease are numerous and varied. These include, rest, corrective shoeing, grooving of the hoof wall, analgesics, warfarin, neurectomy and injection of corticosteroids into the navicular bursa. The number of different treatments is perhaps a reflection of their poor long-term success rate. In 1964 Wintzer published results of different forms of treatment in 580 cases of navicular disease, of which only 4.5% were sound one year later.

Following the findings of Colles and Hickman in 1977, of thrombosis in the distal navicular arteries, and the subsequent hypothesis of ischaemia as a cause of navicular disease, Colles (1979a) initiated a clinical trial using an anticoagulant as a form of treatment. The anticoagulant of choice for this work was the oral dicoumarin derivative, warfarin. Because of the risk of haemorrhage associated with oral warfarin therapy, continual monitoring is required using the one stage prothrombin (OSPT), throughout the period of treatment.

Preliminary results reported by Colles (1979a, 1982) were very promising, particularly if the warfarin was administered in the early stages of the disease before severe fibrocartilage lesions had developed. Since starting to write this thesis another form of drug therapy for navicular disease has been reported by Rose, Allen, Hodgson and Kohnke (1983). This therapy is also aimed at the proposed occlusive vascular nature of the disease process and involves the peripheral vasodilator isoxsuprine hydrochloride. The vasodilator is administered orally for 6-8 weeks, and requires no form of monitoring. Although only a small number of horses have been treated (Rose et al., 1983) this form of therapy has proved very successful.

THE INVESTIGATION

This present study began with an investigation of the clinical features of navicular disease, in order to define the clinical nature of the condition, to examine the incidence of the condition, and to determine whether any predisposition towards the condition was present in the population.

Since navicular disease has not as yet been experimentally produced, clinical examinations were carried out on naturally occurring cases, which were referred to the R.(D.)S.V.S. for confirmation of suspected navicular disease.

An investigation of the radiographic features in the navicular bones of horses in this study was carried out in three parts.

- Part I - An assessment of radiographic techniques in order to find the best method which combined technical ease with the production of consistent diagnostic radiographs.
- Part II - A survey of the radiographic features present in the navicular bones of the horses in this study, in order to establish the radiographic changes associated with navicular disease.
- Part III - The development of an objective method of assessing radiographs of navicular bones using specific radiographic features found in Part II.

A pathological examination of the navicular bones and adjacent portion of the deep flexor tendon from control horses and horses with navicular disease was carried out to assess the pathological changes present in navicular disease, to compare the pathological changes with the clinical and radiographic findings, and to examine the

hypothesis that occlusive vascular disease and local ischaemia was present in the navicular bones of navicular disease cases. The techniques involved in the correlative pathological study included, gross pathology, radiography post-mortem, angiography post-mortem, light microscopy, and fluorescent microscopy following the administration of intra-vital fluorochrome bone labels to a number of horses.

The last part of this study consisted of an evaluation and comparison of different treatments of navicular disease with particular emphasis on oral warfarin therapy. Both clinical and radiographic assessment was made of the results of treatments.

The treatments examined in this study were rest, oral warfarin therapy, corrective shoeing, phenylbutazone, neurectomy, and isoxsuprine hydrochloride.

CHAPTER 2

CLINICAL FEATURES OF NAVICULAR DISEASE

There never was yet a philosopher who could withstand a toothache, but think of a poor horse with 20 toothaches compressed into one agony.

H S DRAKE 1896.

REVIEW OF THE LITERATURE

Probably the first description of navicular disease and its clinical signs in the literature was by Jeremiah Bridges in 1752 although some 2000 years previously the Greeks described what may have been the same condition.

Various descriptions of the clinical condition were published throughout the 19th century (Moorcroft, 1804*; Turner in 1829; Youatt, 1836; Percivall, 1873; Fitzwygram, 1894; Drake, 1896) under various names. The disease was originally called "coffin joint lameness" (Bridges, 1752), and it was later termed "joint capsule lameness" by Sewell (Youatt, 1836). It was Turner in 1829 who first referred to the condition as "navicular joint disease" and one Bracy Clark called it "nut bone disease" in 1839. Percivall in his Hippopathology in 1873 suggested the term "navicular arthritis" was the most suited to the condition, while Brauell (1846) alluded to the disease as "Podotrochlitis".

Navicular disease has also been called "Podotrochleosis" by Olsson in 1954 and "Podotrochlitis Chronica Aseptica" by Numans and Van der Watering in 1973.

The clinical signs of navicular disease are characteristic of a painful condition affecting the posterior part of the foot. The classical signs of the disease are well described by Fitzwygram (1894), Pryer (1934), O'Connor (1946), Hickman (1964) and Adams (1974), although not all of the signs will occur in every case.

It is generally agreed that the typical case has a history of insidious onset of lameness, but as pointed out by Fitzwygram (1894), Pryer (1934), and McCunn (1951), the onset can also be sudden. The condition most commonly affects horses between 7 and 14 years old

* Moorcroft letter to Captain Codrington, 1804, published in The Veterinarian, 1846.

according to Lowe (1974), or 4 to 9 years old according to Ackerman, Johnson and Dorn (1977), although it is by no means confined to these age groups. O'Connor (1946) stated that navicular disease could be found in 2 year olds.

From the surveys of Lowe (1974) and Ackerman et al. (1977) it would appear that the incidence of navicular disease is greater in males than females, but no such sex incidence was found by either Van der Mey, Kleyn and Van der Watering (1967) or by Colles (1982).

It is apparent that no breed or work type of horse is exempt from navicular disease, although many workers feel that definite breed predispositions exist. Law (cited by Drake, 1896), Reeks (1925) and Lowe (1974) considered the thoroughbred to be most often affected, while Rose, Taylor and Steel (1978) noted that in countries such as France where the thoroughbred was seldom raced over 3 years old, navicular disease was uncommonly diagnosed. On the other hand, according to Colles (1982), there is an apparent breed predisposition to the thoroughbred cross horse, and Hickman (1964) maintained that the condition is most frequently encountered in hunters and hacks, and only occasionally reported in the thoroughbred. A British Equine Veterinary Association survey in 1962-63 (BEVA 1965) showed that hunters accounted for 63% of the navicular disease cases recorded. Other workers, Lowe (1974) and Ackerman et al. (1977), considered the Quarter horse to be the most frequently affected while Oxspring (1935) most commonly encountered the disease in polo ponies. It is generally agreed that the condition is rare in heavier breeds and draught horses, although Wirstad (1949) pointed out that navicular disease was very common in the heavy horse of the Norwegian Døle breed. He suggested that this had not been previously noticed

because these horses worked mostly at the walk, and on soft ground, and so the symptoms were often overlooked.

The condition affects the front feet and is usually bilateral, although Rose et al. (1978) reported that of the 70 cases they examined, the presenting lameness was unilateral in 72.9%.

The hind feet can also be affected, and descriptions of the resulting clinical signs are given by Reeks (1925), O'Connor (1946) and Valdez, Adams and Peyton (1978). This is considered by most workers to be rare, and in fact Adams (1974) maintained that the hind feet are never involved unless as a result of puncture wounds.

In the early stages of navicular disease the clinical signs are intermittent and may not be noticed. According to Pryer (1934) and Hickman (1964), the first sign is a loss of action, the horse fails to stride out as before, and has a tendency to stumble. As the disease progresses, this loss of action is followed by slight lameness and pointing of the affected limb or limbs. In contrast Reeks (1925) stated categorically that pointing of the affected limb is the first sign of navicular disease, and this occurs before there is any sign of lameness. Other authors were much in accord with this belief (Brauell, 1846; Percivall, 1873); Drake, 1896; O'Connor, 1946). Despite the difference in opinion as to whether pointing precedes or follows the first signs of lameness, it is unanimously agreed that this action is characteristic of navicular disease, although Pryer (1934) believed that pointing was absent in certain cases.

These early signs are followed by intermittent periods of lameness, which as the disease progresses, become persistent. Percivall (1873) wrote of the intermittent nature of the disease;

... relapse following relapse at intervals, long or short, as the case may be, until, in the end, such morbid changes take place in the diseased foot as render restoration of normal function and feeling impracticable, and the consequence is irremovable lameness.

In the affected foot, in order to reduce weight bearing on the posterior part, the toe strikes the ground first instead of the heels, resulting in a shortened anterior phase of the stride, (Adams, 1974). When both front feet were affected this type of lameness has been described as "grogginess" by Turner (1829) and Percivall (1873), "stubby" by Donahue (1935) and "pottery" by McCunn (1951).

The lameness may be worse when the horse is first taken out of the stable and it then decreases with exercise (Fitzwygram, 1894; O'Connor, 1946; Hickman, 1964; Adams, 1974) but both Percivall (1873) and Pryer (1934) suggested that the degree of lameness was frequently exaggerated by exercise.

Fitzwygram (1894) and McCunn (1951) pointed out that the lameness was increased going downhill. Similarly, O'Connor (1946), Adams (1974) and Johnson (1973) maintained that the lameness was increased when the horse was turned in the direction of the affected foot.

The significance of heat in the affected foot or feet is equivocal. For example Pryer (1934) believed that this symptom was only of value if it was shown to be constant, whereas Hickman (1964) stated of the affected limb; "If the foot is palpated periodically during the course of the day it is found that its temperature does not remain constant but fluctuates, and at times may exhibit considerable heat".

After a period of time the foot gradually changes in shape. Because of lack of use the heels contract, and grow longer, the sole is more concave and the affected foot becomes smaller and more upright.

Such feet are aptly described as "boxy" by O'Connor (1946).

Many of the earlier authors such as Turner (1829), Fitzwygram (1894) and Pryer (1934) were of the opinion that such "boxy" feet were predisposed to navicular disease, while others, (Percivall, 1873; McCunn, 1951; Hickman, 1964) believed that such feet were the result rather than the cause of navicular disease. In the opinion of McCunn (1951); "If a young horse possessed feet of the "blocky" design of the established navicular foot he would most probably never suffer from the disease".

The clinical diagnosis of navicular disease is based upon these clinical signs, although, as mentioned previously, not all of the signs occur in every case, and variations of these can occur (Pryer, 1934).

Clinical diagnosis is aided by various diagnostic tests. Fitzwygram (1894) suggested that pain could be induced by applying pressure on the hollow of the heels, and on the application of concussion to the heels and point of the frog, but that these tests could not be relied on. Reeks (1925) maintained that forcible flexion of the pedal joint sometimes gave rise to tenderness. This could be performed manually, or by placing a wedge of wood under the toe of the affected foot and making the limb bear full weight. A similar test was recommended by Donahue (1935), where, in order to increase the pressure on the navicular bone and deep flexor tendon, the foot was placed on a plank and the front end of this was raised. Wintzer (1964) advocated flexing the fetlock and interdigital joints for one or two minutes and then trotting the horse. He found that 90% of the navicular disease cases showed increased lameness after this.

It is considered that increasing frog pressure will increase the degree of lameness in navicular disease. Several different methods of doing this have been suggested. Reeks (1925) proposed paring the heels in order to leave the frog prominent to take the body weight. The application to the foot, of a bar shoe, produced the same effect, Brauell (cited by Reeks, 1925). The application of a bar shoe was mentioned also by Pryer (1934) as a method of distinguishing navicular disease from pedal osteitis. McCunn (1951) advocated inserting a piece of iron under the shoe at the heels, with a block of wood attached which rests on the frog and then trotting the horse.

The use of hoof testers as a diagnostic aid was suggested by several authors, (Reeks, 1925; Donahue, 1935; Adams, 1974). Pain can be elicited by applying pressure with hoof testers across the middle third of the frog, or across the ends of the navicular bone. As Adams (1974) pointed out, normal horses will show variation in response to hoof testers, so the reaction of the lame foot should be compared with the hindfeet or the opposite fore foot if sound. Rose et al. (1978) found that 72.9% of the cases they examined showed a painful reaction to hoof testers over the middle third of the frog.

It is generally agreed that selective nerve blocks (low palmar digital) play an important part in the clinical diagnosis of navicular disease. These nerve blocks, as Pryer (1934) noted, are not diagnostic of navicular disease, but are used to locate the seat of lameness "to the posterior part of the foot". Wintzer (1964) described local anaesthesia of the palmar digital nerves and of the pedal joint as "the most useful diagnostic aid". He found that after palmar digital nerve block in 393 horses, 91.6% of the animals

went sound, or lame in the other foreleg, and after local anaesthesia of the pedal joint in 143 horses, 88.8% were sound. No significant difference was found between these two methods of anaesthesia. If the site of lameness had been located to the area innervated by the palmar digital nerves, in the posterior third of the foot, but doubt existed as to whether the cause of lameness was in the navicular apparatus or other structures in the area, then Schebitz (1964) recommended injecting local anaesthesia into the pedal joint. The local anaesthetic, he maintained, would diffuse from there into the navicular apparatus, but not affect other structures in the heel region, and so help to clarify the situation.

Colles (1982) reported that in 93% of his navicular disease cases, there was increased pulsation and diameter of the digital arteries in at least one foot, and although a non specific finding, it is a useful guide in diagnosing the condition. Turner (1829) also considered this an important criterion in diagnosing navicular disease. Smith (1886) however stated categorically that he did not "believe in throbbing of the plantar arteries", in navicular disease. Colles (1982) concluded that there was no specific test for the diagnosis of navicular disease, but that the diagnosis was based on the response to a number of clinical tests and then confirmed by nerve block.

The radiographic examination of the navicular bone is an essential aid in the diagnosis of navicular disease and is discussed in Chapter 3.

MATERIALS AND METHODS

HORSES

Clinical examinations of the locomotor system of 265 horses were carried out in a three and a half year period from 1979-1983. Of these 265 horses, 90 constituted a control population (Group A). The control horses and ponies were referred to the R.(D.)S.V.S. Veterinary Field Station for conditions other than lameness. There was no history of lameness, and no evidence of lameness on clinical examination. The clinical details of these animals are contained in APPENDIX 2.1.

The remaining 175 horses were referred to the Department of Surgery for confirmation of suspected navicular disease.

CLINICAL DIAGNOSIS OF NAVICULAR DISEASE

The clinical criteria used for the diagnosis of navicular disease were:

- (i) A history of forelimb lameness, usually, but not always, of insidious onset.
- (ii) A lameness which was either unilateral or bilateral.
- (iii) A shortening of the anterior phase of the stride, the toe of the foot contacting the ground before the heels, during movement. As a result of this there may be marked wearing of the toe of the shoes, and often a history of stumbling with one or other front foot.
- (iv) A lameness which was often marked when the horse was turned in the direction of the affected limb or limbs.

- (v) Pointing of the affected limb or limbs when at rest.

Pointing was considered to be present when the affected limb was either held out almost fully extended, the foot usually flat on the ground, or held out only a short distance in front of its normal position with the toe of the foot on the ground, the heels slightly raised off the ground and the fetlock joint flexed forward. The horse may alternate which foot it pointed or it may point both front feet at the same time, to produce an almost "rocking horse" stance.

- (vi) Alteration in the shape of the affected foot or feet - the foot becomes smaller and narrower and higher at the heels.

- (vii) That no difference in the degree of lameness could be detected when the horse was trotted on soft or hard ground.

- (viii) Detection of heat in the foot or in the heels of the foot.

Aids to Diagnosis

The following tests were used in the clinical examination of these horses, and their value as aids to the diagnosis of navicular disease was assessed.

- (a) Pressure with hoof testers was applied across the heels and across the middle third of the frog (Adams, 1974).

The hoof testers were applied to the sound forefoot first when the lameness was unilateral, in order to assess the response more clearly. A positive result was recorded to this test if the horse showed a pain reaction either by a change in expression and/or retraction of the limb.

- (b) Hyperflexion of the pedal joint was carried out on both front feet. A positive result was recorded if the horse showed a pain reaction as above.

- (c) Following flexion of the pedal joint for one to two minutes, the horse was trotted - a positive result was indicated by an increase in the degree of lameness for a few strides following flexion (Wintzer, 1964).

The results of both (b) and (c) were combined to give the flexion test, and a positive reaction to one or both parts was recorded as a positive flexion test.

- (d) Low palmar digital nerve block was performed. It was not possible to carry out this diagnostic procedure in all cases, and of the 175 lame horses 158 of them received a low palmar digital nerve block. When the lameness was bilateral the worse affected leg was blocked. The nerve block was performed in each case with the leg raised, and after suitably preparing the sites on the medial and lateral aspects of the pastern, a 23 gauge 1 inch needle was inserted subcutaneously at right angles to the long axis of the pastern, slightly anterior to the edge of the deep flexor tendon and $\frac{1}{2}$ an inch above the lateral cartilages. One to two millilitres of 2% lignocaine hydrochloride* were then injected at each site. The efficacy of the nerve block was tested by loss of skin sensation over the posterior third of the coronary region, before its effect on lameness was assessed. A positive result was recorded when the lameness was relieved or improved following low palmar digital nerve block in that limb.

Clinical examinations and diagnoses were carried out prior to any radiographic examination of the navicular bones.

* Xylocaine, Astra Pharmaceuticals Ltd, Watford.

The clinical details of the 265 horses examined were used to investigate the incidence of navicular disease and to determine whether any predisposition towards the condition existed in the population. Differences in age between the groups of horses was assessed by analysis of variance (TABLE 2.1). Differences in incidence of clinical findings were tested for significance against chi-squared distribution (TABLE 2.2, 2.3). The details and results of clinical examination of the 175 lame horses referred to the Department of Surgery are contained in APPENDICES 2.2 and 2.3.

DEGREE OF LAMENESS

The assessment of the degree of lameness shown by an animal is a highly subjective process, and the standardisation of such assessment very difficult (Silver, 1982). It was nevertheless considered necessary in this work to have some method of quantifying the degree of lameness present in each case.

A four grade system was used for this purpose, and in order to maintain a degree of consistency, grading of the degree of lameness in each case was carried out by one person.

Grade 1 - This was defined as a very slight lameness, apparent either as a shortening of the stride or failure to stride out properly, with lameness only becoming noticeable when the horse turned in the direction of the affected limb or limbs, or, as a mild supporting leg lameness which was not usually evident with every stride.

Grade 2 - This was defined as a slight to moderate degree of supporting, leg lameness, present with every stride, but not pronounced, and often only evident in one limb.

Grade 3 - This was a much more pronounced supporting leg lameness than that of Grade 2. In this case it was very often bilateral, with one limb showing more severe lameness than the other.

Grade 4 - This was defined as a severe lameness, usually bilateral, accompanied by a very much shortened stride, giving a "paddling" appearance to the gait. With this degree of lameness, marked discomfort was usually present at rest, the horse continually shifting weight from one front foot to the other.

The degree of lameness was assessed in all horses at the time of clinical examination, before radiographic examination had been carried out.

RESULTS

Of the 175 horses referred as possible navicular disease cases, 151 of them had a lameness which was diagnosed according to the clinical criteria set out previously as navicular disease (Group B) APPENDIX 2.2.

The other 24 horses have been designated the "not yet diagnosed" (NYD) group. These were horses with foreleg lameness which was located in the foot or feet, but which fulfilled less than 50% of the clinical criteria for navicular disease, and could not be diagnosed as any specific disease entity (Group C) APPENDIX 2.3.

INCIDENCE AND PREDISPOSITION

No significant sex or colour incidence was found between the control group, the navicular group and the NYD group of horses. A significantly high incidence of navicular disease was recorded in the thoroughbred cross horse, but no other breed incidence was apparent. A significant age distribution was noted between the control horses and both groups of lame horses, although no difference in age distribution was found between the navicular disease groups and the NYD group of horses. The age of the control horses ranged from 1 year old to 16 years old, with a peak at 7 years old. The age of the navicular disease group ranged from 4 years old to 18 years old, with the maximum incidence occurring at 9 years old. In the NYD group the age of the horses ranged from 5 to 16 years old, with a peak incidence at 8 years old (FIG 2.1).

The type of work carried out by the horse did not appear to be related to the incidence of navicular disease, and all work types are represented in the navicular disease group.

CLINICAL EXAMINATION

The duration of lameness before referral was recorded, although in some cases this was not known, since the animal had only been recently purchased or had not been in use for several months. In the navicular disease group the duration of lameness ranged from 2 weeks to 3 years.

The lameness was of insidious onset, the early history being one of intermittent bouts of lameness, in 84% of the navicular disease cases. The other 16% of cases had a sudden onset of lameness with no previous history of forefoot lameness.

In the NYD group the duration of lameness ranged from 2 weeks to 2 years, and was of insidious onset in 13(54%) horses and of sudden onset in 11(46%) horses.

Lameness was bilateral in 117(77.5%) of the navicular disease cases and 8(33.3%) of the NYD cases, and unilateral in 34(22.5%) of the navicular disease cases and 16(66.6%) of the NYD cases.

From clinical examination it was found that 146(96.7%) of the horses with navicular disease showed shortening of the stride, the toe of the foot contacting the ground before the heels. This action was also recorded in 12(50%) of the horses in the NYD group, but was not recorded in any of the control horses. An increase in the degree of lameness when the horse was turned in the direction of the affected limb or limbs was found in 131(86.7%) of the navicular disease group, in 17(70.8%) of the NYD group but again was not a feature in the control horses. Stumbling or tripping with one or other front foot occurred in 9(10%) of the control horses and in 5(20.8%) of the horses in the NYD group, but had a highly significant incidence in navicular disease group, 74(49.9%).

The action of "pointing" when at rest, was not present in all of the navicular disease cases. It was noted in 135(89.4%) of them and in 10(41.6%) of the NYD group. Pointing also occurred in 2 of the normal control horses. Seven of the 135 horses with navicular disease found to point, pointed each front foot alternately, and 21 of the 135 horses pointed both front feet at the same time.

Changes in foot conformation, the foot becoming smaller, narrower, and higher at the heels, was recorded in one foot in 121(80.1%) of the horses with navicular disease, and both front feet were affected in 18(11.9%) of the cases. Ten horses in the NYD group showed a change in the shape of one front foot typical of navicular disease, and 1 horse had changes in both front feet. In the control group of horses one foot was found to be smaller and narrower and higher at the heels, than the other, in 13(14.4%) cases.

It was decided to record the number of horses with bruising of the soles of one or both front feet. This was present in 9(10%) horses from the control group, 4(16.6%) horses from the NYD group and in a significantly higher number 61(40.4%) from the navicular disease group.

AIDS TO THE DIAGNOSIS OF NAVICULAR DISEASE

In the navicular disease group all three aids to diagnosis were used in 130 horses. Of these horses 32(24.6%) showed a positive reaction to all three; 84(64.6%) showed a positive reaction to two of them and 14(10.8%) showed a positive response to only one. No horse in the navicular disease group showed a negative reaction to all three diagnostic aids.

Pressure with hoof testers was used as an aid to diagnosis in all horses examined. In the navicular disease group, results were recorded in only 148 of the cases, because 3 horses were fractious and a true reaction could not be assessed. Of the 148 horses, 67 (45.2%) gave a positive response to pressure with hoof testers. Pressure with hoof testers was applied in the same manner to the 90 control horses, and a positive response was shown by 5 (5.5%) of them.

The flexion test, as described earlier, elicited a positive reaction from 110 (72.8%) of the horses in the navicular disease group, and following hyperflexion of the pedal joint a positive response was shown by 5 (5.5%) of the 90 control horses.

Low palmar digital nerve block was performed in 133 of the horses in the navicular disease group. Either relief from lameness, or improvement in lameness, occurred in 129 (96.2%) cases. In 5 cases no response to the nerve block was recorded, although there was loss of cutaneous sensation around the posterior third of the foot. Four of these 5 horses had shown a positive reaction to both hoof testers and to the flexion test. In the navicular disease cases with bilateral front foot lameness, following low palmar digital nerve block of the worse affected foot, 56% of them showed distinct lameness in the opposite foreleg.

In the NYD group no horse showed a positive response to all three aids to diagnosis and 8 (33.3%) showed no response to any. In this group a positive response to pressure with hoof testers was recorded in 6 (25%) horses, to the flexion test in 9 (37.5%) and to low palmar digital nerve block in only 6 (25%). There was a significantly higher response to the flexion test and to low palmar digital nerve

block in the navicular disease group of horses than in the NYD group, although no significant difference was found between the two groups in response to pressure with hoof testers.

DEGREE OF LAMENESS

Grade 1 - Lameness was considered to be present in 20(13.2%) of the horses with navicular disease, and in 8(33.3%) of the horses in the NYD group.

Grade 2 - Lameness was recorded in 62(41%) horses in the navicular disease group and in 10(41.6%) of the horses in the NYD group.

Grade 3 - Lameness was present in 56(37%) of the horses in the navicular disease group and in 5(20.8%) of the horses in the NYD group.

Grade 4 - Lameness was recorded in 13(8.6%) of the horses with navicular disease and only 1(4.2%) horse from the NYD group.

No significant difference was found in the occurrence of grade 2, 3 and 4 lameness between the navicular disease, and the NYD groups of horses, while grade 1 lameness was found at a significantly higher level in the NYD group than in the navicular disease group.

When the duration of lameness obtained from the case histories was related to the grade of lameness as assessed subjectively, in each horse, a significant positive correlation was found in the navicular disease group (FIG 2.2). No significant correlation was found in the NYD group (FIG 2.3).

During clinical examination of the navicular disease group of horses, 7 were found to be lame in the hind limbs as well as lame in the front limbs. APPENDIX 2.4. Three of these were found to be lame on both hind limbs and the others on only one hind limb. Clinically these horses showed shortening of the anterior phase of the stride, the toe of the foot contacting the ground first, and an increase in the degree of lameness when turned in the direction of the affected limb or limbs. Only 3 of the horses showed a positive response to pressure with hoof testers, but all of them gave a positive reaction to the flexion test. In the 4 cases in which a low plantar nerve block was performed, a positive response was recorded.

The hind limb lameness in these cases was diagnosed clinically as navicular disease, and this was later confirmed by radiography.

DISCUSSION

Information obtained from horse owners, case histories, clinical examination, and the assessment of the degree of lameness are all highly subjective processes. This problem is accentuated by the fact that a horse, in its lifetime, frequently changes both ownership and purpose for which used, often on several occasions. However, it was considered important from the point of view of this work that such information should be recorded, more as a useful guide or indicator to the clinical features of navicular disease, rather than for the purpose of statistical analysis.

The 265 horses examined during the course of this work cannot be said to represent a random population, since a random population is defined as one in which "each member of the population has an equal probability of being selected" (Collins, 1979). However, since the lame horses were all referred to the R.(D.)S.V.S. Veterinary Field Station for confirmation of navicular disease, and since the control horses used in this work were also referred cases, and so therefore the same bias must apply, the groups were considered to be comparable.

All clinical examinations and diagnoses based upon the clinical examination in the horses used in this work were carried out prior to any radiographic examinations of the navicular bone. Although it is generally accepted that radiography of the navicular bone is a very useful aid to diagnosis of navicular disease, it was decided to base the diagnosis of the condition initially upon the results of the clinical examination, so that the results of the radiographic examination did not in any way influence the diagnosis, and so that true significance of the radiographic findings could be assessed separately.

Navicular disease was not confirmed in all 175 of the horses referred to the Department of Surgery. Twenty-four of these horses, although they showed some clinical features of navicular disease, could not be diagnosed as such nor as any other specific disease entity, and so for the purpose of this work these horses were classed as the "not yet diagnosed" group (Group C). Although the numbers in this group were small, their clinical details have been included for comparison with those of the confirmed cases of navicular disease and of the control horses.

From this study it can be seen that a high incidence of navicular disease was recorded in the thoroughbred-cross horse, thus confirming the findings of Colles (1982). It is difficult to know how much significance to attach to this finding since the thoroughbred-cross horse is not a clearly defined entity, and can vary, from half-thoroughbred, threequarter-thoroughbred, seveneighths-thoroughbred to combinations thereof.

When the type of work carried out by thoroughbred-cross horses was examined, it was found that there was a significantly high percentage of thoroughbred-cross horses used for eventing, but this was true of both the navicular disease group of horses and control group of horses. The apparently high incidence of navicular disease in the thoroughbred-cross horses in this study is therefore not related to the type of work performed. It has been suggested that an unknown predisposing factor may be present in the thoroughbred-cross horse but not in the pure bred (Colles, 1982). On the other hand, there may be many factors involved such as frequency of work, different training programmes, foot conformation and shoeing. McCunn (1951) believed that the type of foot most susceptible to

navicular disease was the flat weak foot, with long toes, low heels and flat soles. This, he considered, was most frequently encountered in the "half or threequarter-bred hunter". Colles, Garner and Coffman (1979) showed that, in a foot with this conformation, there was a considerable slowing of the rate of blood flow through the foot which may predispose it to navicular disease. When the horses in this present work were examined for long toe, low heels conformation it was found that 33(21.8%) of the horses in the navicular disease group were affected, and of the 33 horses, 22(66.6%), a significantly high percentage of them were thoroughbred-crosses. In the control group of horses 9(10%) had long toe, low heel conformation and of these 4(44.4%) were thoroughbred crosses. It is suggested therefore that one of the factors involved in the high incidence of navicular disease in the thoroughbred cross horse may be the high incidence of long toe, low heel conformation in these animals.

A significant age distribution was found in the navicular disease group which did not differ markedly from that of the NYD group, the peak occurrences being at 9 years old and 8 years old respectively. No horse in this study, less than 4 years old, had navicular disease, although according to O'Connor (1946) the disease occurred in 2 year old horses. It is likely that the age distribution of navicular disease is related to the period of time when the greatest work output and maximum fitness is demanded of these animals, and indeed navicular disease has often been described as affecting horses "in their prime of life" (Hickman, 1964). It is also likely that during this period of hard work horses are more prone to sustain injuries, orthopaedic or otherwise, and as a result of these, sudden and sometimes prolonged rest will be required. From an examination of the

histories of the navicular disease cases in this work, there is no doubt that, in a large number of them, the onset of the clinical signs of navicular disease occurred following a period of rest, either an elective period of rest as in horses used for hunting and eventing, or a period of rest due to injury and illness. It is interesting to note that the clinical signs in these cases usually occur at the time, or shortly after, the horse is returned to work. These observations are in accord with those of Pryer (1934) and Colles (1982).

Miles in 1846 stressed the importance of regular daily exercise, and the housing of horses in loose boxes instead of stalls, to allow movement, for the prevention of navicular disease.

In this study it was found that the incidence of navicular disease was not related to any particular type of work performed by the horse. Other workers have found a high incidence of navicular disease in horses used for hunting (Hickman, 1964; Colles, 1982) and polo ponies (Oxspring, 1935). The importance of the specific type of work performed by the horse as regards predisposing it to navicular disease cannot be accurately estimated, since this factor is very variable, and may only account for the type of work being carried out at the time of the onset of the clinical signs. Most horses, by the time they reach the age of 9 years old, the age of maximum incidence of navicular disease, have passed through several different work categories. It may have been more useful, but perhaps impracticable, to have examined the complete history as regards different types of work and management of these horses.

The majority of the navicular disease cases had the typical history of gradual onset of lameness, often only realised in retrospect. This did not apply in all cases, and the sudden onset of

often quite severe lameness should not preclude a diagnosis of navicular disease.

The duration of lameness before referral was recorded, and although it could not be guaranteed to be accurate, it was thought to be a useful indicator.

Bilateral forelimb lameness was found more often (77.5%) than unilateral forelimb lameness in the navicular disease cases. This was often difficult to detect, particularly when the lameness was only slight, and both front feet were affected to the same degree.

From clinical examination of the three groups of horses the clinical features which are considered to be of particular significance in the diagnosis of navicular disease are:

- (i) The gait - in 96.7% of the confirmed cases of navicular disease there was distinct shortening of the stride, the toe of the foot contacting the ground first before the heels. Although this action is only indicative of a painful lesion in the posterior part of the foot, it was present in virtually all the cases of navicular disease examined, and was considered to be a characteristic clinical feature. The 5 cases in which this characteristic gait was not present all showed a shortening of the stride, but the heel of the foot was placed on the ground first. Two of the 5 cases had a recent history of laminitis, and the other 3 had marked bruising of the sole at the toe in both front feet.
- (ii) An increase in lameness when the horse was turned in the direction of the affected limb or limbs. This was present in 86.7% of the navicular disease cases and also in 70.8% of the NYD cases. Although a non-specific finding, nevertheless it was

considered to be a useful method of detecting bilateral lameness, particularly when both front feet were affected to approximately the same degree.

- (iii) Pointing - as Pryer (1934) had suggested, this feature was not present in all the cases of navicular disease. The degree to which the horse pointed the affected foot, whether it was far in advance of its normal position or only slightly in advance, did not appear to be related to the degree of lameness. However, characteristically in severe cases of navicular disease, bilateral pointing or "rocking horse" stance was present (FIG 2.4). This stance was recorded in 21 of the navicular disease cases, and all but one of them had grade 3 or 4 lameness. It was not found in any of the control horses or in the NYD group of horses.

This stance was therefore considered to be a very useful indicator of advanced bilateral navicular disease.

Other clinical features worthy of note include:

- (i) Alteration in hoof conformation as described earlier. This was found in 92% of the navicular disease cases, and, in some of these, both front feet were affected. It was also found in 14.4% of the control horses in only one front foot. It is possible that this difference in foot shape, in these control horses, could have arisen as a result of some sub-clinical condition resulting in favouring of one foot, or as a result of some conformational abnormality. It is therefore considered that too much significance should not be placed on the change in shape of one front foot, in the absence of other clinical findings.

Waisting of the hoof wall was noted in the front feet of a number of navicular disease cases. These navicular disease cases were usually of between 4 and 6 months duration.

Waisting took the form of a groove in the hoof wall, about half way between the coronary band and the ground surface, which started at the heels and ran forward.

- (ii) Heat - detected in the foot, and/or in the bulbs of the heels of the foot, in many of the navicular disease group of horses and also in the NYD group. Unfortunately, as Hickman (1964) pointed out, this was not a constant finding and varied throughout the day and from day to day. This finding was therefore not recorded in the clinical details.

AIDS TO THE DIAGNOSIS OF NAVICULAR DISEASE

Of the aids to diagnosis used in the clinical examination, low palmar digital nerve block gave the most consistent results. In only 5 of the navicular disease cases blocked there was no response. In these 5 cases most of the other clinical criteria were positive, and for this reason they were diagnosed as navicular disease cases, despite the negative response to low palmar digital nerve block. The failure of these horses to respond positively to the nerve block could be due to a number of reasons:

- (i) Failure to produce anaesthesia of the navicular apparatus, despite the loss of cutaneous sensation in the posterior third of the foot. This may be brought about by the existence of so called accessory nerve branches (Adams, 1974), which arise from the palmar digital nerve, or from the digital nerve, to supply the navicular area.

- (ii) Extensive bruising of the foot in the region of the toe.

In the characteristic navicular disease gait, the toe of the foot contacts the ground first, and this can lead to bruising of the toe area. So, despite having removed sensation from the navicular area, the horse would still remain lame.

- (iii) Mechanical reasons - such as extensive adhesions between the navicular bone and the deep flexor tendon. The horse would then be unable to alter its gait to any extent, despite the nerve block.
- (iv) The lameness being the result of conditions other than navicular disease.

In the 5 navicular disease cases showing a negative response to low palmar digital nerve block, a high palmar nerve block was performed. Three of the horses showed a positive response to this, the remaining 2 showing virtually no change in their gait. Of the 3 horses responding positively to the high block, 2 were found to have marked bruising at the toes of both front feet, and this was thought to be the reason for failure to respond to the low nerve block. In the remaining horse, no reasons other than navicular disease could be found for the lameness, and so its failure to respond to the low nerve block may have been due to accessory nerve branches. Post mortem examination was carried out on the 2 horses that had failed to respond to either the low or high nerve blocks. In one horse extensive adhesions were found between the navicular bone and the deep flexor tendon in both front feet, and in the other horse, almost complete rupture of the deep flexor tendon was found, at the level of the navicular bone, in one front foot. These lesions were considered to be the cause of the negative response to low palmar digital nerve block.

The flexion test also proved a useful diagnostic aid, giving 72.8% positive response in the navicular disease cases. In some horses the response to hyper-flexion of the pedal joint was not entirely clear, but if the joint was held in a normal degree of flexion for one to two minutes, and then the horse trotted, these horses invariably showed an increase in lameness for the first few strides. Wintzer (1964) found this latter test to be positive in 90% of the navicular disease cases he examined.

It is therefore advisable that both parts of the test are performed before a response is recorded.

The application of pressure with hoof testers results in fewer positive responses than did the other two diagnostic aids. The response was found to vary, depending upon the user and the amount of pressure applied. Like Adams (1974), more response was recorded when pressure was applied with hoof testers across the middle third of the frog than when applied across the end of the navicular bone. In the navicular disease group of horses, 45% showed a positive response to hoof testers. This figure is far less than the 100% response quoted by Rose et al. (1978). Because of the variability in response depending upon the user, amount of pressure, and even type of hoof testers, this test was not considered to be as useful a diagnostic aid for navicular disease as the flexion test.

ASSESSMENT OF THE DEGREE OF LAMENESS

This process was complicated, particularly in the grade 1 and 2 lameness, by variations in the degree of lameness from day to day. The lameness in some cases would improve following exercise, and then be worse the next day. This was taken into account as much as

possible, although this variation was not generally a feature of the grade 3 and 4 lamenesses.

A positive correlation was found between the degree of lameness based on the grading system used, and the duration of lameness. In other words, the longer the duration of lameness, the more severely lame the horse tended to be. This correlation would tend to confirm the progressive nature of navicular disease. No such correlation was present in the NYD group of horses. More low grade (1 & 2) lameness was present in this group.

Hickman (1964) described navicular disease cases, in 11-15 year old horses, in which there was slight intermittent lameness which did not rapidly get worse. These he considered were the result of different pathological lesions affecting the navicular bone. It is possible that some of the lameness in the NYD group could be the result of a low grade, non-progressive or only slowly progressive form of navicular disease, although they did not fit the clinical criteria.

The occurrence of navicular disease in the hind feet has rarely been reported (Reeks, 1925; O'Connor, 1946; Valdez et al., 1978). From clinical examinations of the 265 horses used in this work, 7 were found to be lame in the hind feet, and the clinical signs and response to diagnostic aids were suggestive of navicular disease. This was later confirmed by radiography, and in 4 cases by nerve block. Six of these 7 horses were referred for front limb lameness, the hind limb lameness going unnoticed. Only one case was referred with a hind limb lameness, as well as fore limb lameness. All the horses examined with navicular disease in the hind feet also had navicular disease in the fore feet. The presenting clinical signs

of hind limb navicular disease were similar to those found in fore limb navicular disease, except for 2 features. Stumbling was not noted in any of these cases, and instead of pointing the affected limb, or limbs, as in fore limb lameness, the hind limb was rested with the toe of the foot just touching the ground.

Both the flexion test and low palmar digital nerve block proved particularly useful diagnostic aids in hind limb navicular disease.

It is possible that navicular disease affecting the hind feet is not as rare as has been previously thought, and that the detection of this is obscured by the more obvious fore limb lameness, particularly if the fore limb lameness is bilateral and severe.

CONCLUSIONS

It has been said of navicular disease that "no form of lameness is more mis-diagnosed" (Dollar (cited by Pryer, 1934) and that it is "the last hope of the diagnostically destitute" (Rose et al., 1978). Pryer in 1934 suggested that mis-diagnosis of the condition was due to the fact that, having described the "classical" signs of navicular disease, authors then went on to describe all the variations thereof, thus leaving the clinician wondering which clinical features were to be regarded as important in the diagnosis.

From this work it was concluded that no one clinical feature was diagnostic of navicular disease in itself, but that the clinical diagnosis should be based upon the presence of a number of clinical signs, and, the response to a number of diagnostic aids. This confirms the findings of Colles (1982).

Features which were considered to be of particular significance in confirming the clinical diagnosis of navicular disease were:

- (i) The presence of the characteristic gait - shortening of the stride, the toe of the foot contacting the ground first. This is considered to be the most important of the clinical signs.
- (ii) The presence of pointing. Bilateral pointing of the front limbs - "rocking horse stance", proved to be a very good indicator of severe or advanced navicular disease.
- (iii) A positive response to flexion test, although a non specific test, is a good aid for confirming clinical suspicions of navicular disease. A negative response to the flexion test does not preclude a diagnosis of navicular disease.
- (iv) Low palmar digital nerve block is considered to be very important in confirming the diagnosis of navicular disease.

A positive response to nerve block, in the presence of other clinical signs, will confirm a diagnosis of navicular disease, but a negative response in the presence of a number of other clinical signs should not rule out a diagnosis of navicular disease.

It is concluded that navicular disease affecting the hind feet may be more common than has been previously thought. As this appears to occur in addition to the front feet being affected, its importance per se is questionable, but it may significantly affect the prognosis.

Lastly, it would appear from this study that the particular type of work performed by a horse does not, in itself, predispose the animal to navicular disease, but rather the common factor present in a large number of the navicular disease cases was a history of irregular work, and sudden or prolonged periods of rest in an otherwise hard worked horse. This factor, along with others such as hoof conformation and shoeing, may render the horse more susceptible to navicular disease.

CHAPTER 3

RADIOGRAPHY OF THE NAVICULAR BONE

Resemblances are the shadows of differences. Different people see different similarities and similar differences.

Vladimir Nabokov (1962)

REVIEW OF LITERATURE

The first report of radiography of the navicular bone as a diagnostic technique was by Eberlein in 1913 (cited by Olsson, 1954). He found radiographic changes in the navicular bones of 8 horses suffering from navicular disease, using a latero-medial projection. It was at least 20 years after this before radiographic examination was to become an important factor in the diagnosis of navicular disease, mostly due to technical difficulties.

In 1931 and 1935 two important works were published on radiography of the navicular bone by Pryer and Oxspring respectively. Of the navicular bone Pryer (1931a) wrote;

The navicular bone affords a curious problem to the x-ray examiner. It is a small bone say two and a quarter inches in length and three-quarter of an inch in width, situated deeply within the horse's hoof, and radiological access to it is through the much thicker os coronae, a position comparable in some ways possibly to a postero-anterior of the human patella.

He then went on to describe the technique he employed, which was to become known as the "high coronary route".

Oxspring (1935) described, in detail, the radiographic techniques involved and the radiological diagnosis of navicular disease. He recommended for routine radiographic diagnosis an antero-posterior view of the navicular bone using the "upright pedal route". This "upright pedal route" he found to be superior to the "high coronary route", (Pryer, 1931a) and to the latero-medial projection.

Because of the anatomical position of the navicular bone on the posterior aspect of the pedal joint, in order to obtain a clear antero-posterior view of the navicular bone, either the angle of the x-ray beam must be carefully controlled in order to project the distal border of the bone above the shadow of the pedal joint, as in the

"high coronary route", or the position of the navicular bone must be altered as in the "upright pedal route".

In the "high coronary route" described by Pryer in 1931(a), the x-ray film was held in a reinforced aluminium cassette which was placed on the ground, the horse's foot was then positioned on the cassette and the x-ray beam, angled at 60° to the horizontal, was centred on the middle of the coronary band. In the "upright pedal route" described by Oxspring (1936), the foot was positioned in a stand such that the anterior wall of the hoof was at right angles to the ground, and the x-ray film in a cassette was placed behind the heels. When the foot was in this position the navicular bone lay parallel to the film (Oxspring, 1935). The x-ray beam is again centred on the middle of the coronary band. With this latter positioning there is a sizeable airspace between the foot and the film, resulting in a large amount of scattered radiation. This scattered or secondary radiation greatly reduces image definition. It was not until the introduction of the Lysholm grid, to reduce scattered radiation, by Oxspring in 1935, that the "upright pedal route" became of value.

Numerous workers have written on the advantages and disadvantages of each of these techniques. Olsson (1954) favoured the "high coronary route", the foot being placed on a reinforced cassette and the x-ray beam at the greatest possible angle of 80° to the horizontal. He pointed out that an angle of 50° - 60° would result in a distortion of the navicular bone image. Carlson (1977) recommended a slight variation of this route, the toe of the foot being raised on a small block, with the x-ray beam at an angle of 60° to the horizontal, centred on the second phalanx.

A comparative study of the two routes was carried out by Campbell and Lee in 1972. They concluded on the basis of their results, that the "high coronary route" with the x-ray beam angled at 60° to the horizontal was the most satisfactory technique, both in technical ease and in the radiographs produced.

The "upright pedal route" was recommended by Douglas and Williamson (1963). They suggested that two antero-posterior views of the navicular bone should be taken, one with the front of the hoof wall at right angles to the horizontal in order to obtain a clear view of the proximal border, and the other with the hoof wall at 80° to the horizontal to obtain a clear view of the distal border. Also in favour of the "upright pedal route" were Hickman (1964) and Schebitz and Wilkens (1978).

Colles (1982) maintained that the picture quality was very similar using the two techniques, but that the "upright pedal route" was technically superior to the "high coronary route". The latter resulted in a degree of navicular shadow lengthening, although this did not seriously affect interpretation.

The use of a dorso-volar projection of the navicular bone was advocated by Morgan (1972) O'Brien, Millman, Pool and Suter (1975); Rose et al. (1978) and Deppe, Tellhelm and Leppert (1981). In this projection the foot was placed on a reinforced cassette and the x-ray beam centred downwards and forwards between the bulbs of the heels at an angle of 30° to the ground, or according to Deppe et al. (1981) 45° to the ground. The promoters of this projection maintained that it demonstrated the important tendinous surface better than the other views. On the other hand, Colles (1982) found that the dorso-volar view gave little additional information over the

antero-posterior views, and that the technique was more difficult to carry out.

Dünnemann (1937) considered the latero-medial projection of the navicular bone to be of little diagnostic value, and Oxspring (1935) maintained that lateral radiographs of the navicular bone were not suitable for diagnosing early cases of navicular disease, but had proved valuable in confirming advanced cases. Morgan (1972) believed that the lateral view of the navicular bone had "not received the attention it deserves", and he stressed the importance of producing a perfect lateral projection to avoid misinterpretation. Colles (1982) also emphasised the importance of the lateral view of the navicular bone in the diagnosis of navicular disease. From his pathological survey he reported that damage to the tendinous surface of the bone could generally be detected on a lateral view.

It was pointed out by both Pryer (1931) and Oxspring (1935) that in order to obtain a radiograph of the navicular bone, the x-rays must pass through the solar surface of the foot, which is very irregular in its thickness and density. As a result of this the shadows of the lateral and central clefts of the frog would overly the navicular bone and could lead to misinterpretations. Pryer (1931) resolved this problem by packing these clefts with a mixture of fine hoof raspings and lard. This mixture appeared to be of roughly the same radiographic density as the horn of the sole. Since then it has become accepted practice, before radiography of the navicular bone, to remove the shoe, clean and pare the foot, and pack the clefts of the frog with a suitable packing material. The materials most commonly used are, soft soap, play-doh*, and vaseline, (Douglas and Williamson, 1980; Colles, 1982).

* The Palitoy Company, Leicester, England.

Adams (1974) maintained that the best diagnostic radiographs were taken with the foot placed in a water bath in order to eliminate the shadows of the frog clefts. In contrast, Douglas and Williamson (1970) suggested that most radiographers preferred not to fill the clefts of the frog, in order to recognise them and then allow for this in the interpretation.

Considerable difference in opinion exists regarding the radiographic changes and their significance in navicular disease. Oxspring (1935) believed that navicular disease could be diagnosed radiographically before it had become clinically apparent. Wintzer (1964) agreed with this finding. Rose et al. (1978) and Colles (1982) considered that radiographic changes were present in all cases of navicular disease. On the other hand Westhues (1938) along with other workers (Olsson, 1954; Carlson, 1967; Adams, 1974; Ackerman et al., 1977) maintained that in many cases of navicular disease no radiographic changes could be demonstrated.

Pryer (1931) commented that he had found "very striking differences" in the radiographic appearance of navicular bones from normal horses and those with navicular disease. The first radiographic lesions to be demonstrated by Pryer in 1931 were, an increased width of the navicular bone - the commonest lesion, and bony exostoses on the proximal border of the medial and lateral wings of the navicular bone - or "spurs". "Spurs" he considered to be a more advanced change. Later, in 1934, Pryer succeeded in demonstrating carious areas in the navicular bones of some horses suffering from navicular disease.

Oxspring (1935) described the earliest radiographic changes of navicular disease as a decalcification in the centre of the bones, and in some cases around the nutrient foramina in the distal border, giving

them a larger and more irregular appearance. These he wrote "give prima facie evidence of the onset of disease".

As the disease advanced he noted the development of caries within the bone which were "quite unmistakable". Oxspring explained that although increased width of the navicular bone could occasionally occur as a sequel to navicular disease, the width of the bone varied with the position of the x-ray beam relative to the bone. Dünemann (1937) agreed with Oxsprings' findings in essence, but considered that these changes were found only in advanced cases of navicular disease.

Although larger and more irregular nutrient foramina in the distal border of the navicular bone were found in well established cases of navicular disease, Westhues (1938) observed that larger foramina than normal were to be found in very old horses without lameness, and this therefore could not be taken as evidence of navicular disease. Numans and Van der Watering (1973) also considered that age affected the development of the nutrient foramina, and that foramina were frequently absent in normal horses up to three years old. Changes in the nutrient foramina in young horses were, they concluded, more significant than similar changes in older horses. Colles and Hickman (1977) reported that the size of the normal nutrient foramina in the distal border of the bone was not related to age, but to the type and frequency of the work performed.

Olsson (1954) from radiographic examination of macerated navicular bones, reported that the earliest change demonstrable in navicular disease was an increase in vascularisation. This was evident as an increase in the size of the nutrient foramina in the distal border of the navicular bone and, in some cases, the appearance of foramina in the proximal border of the bone. Later changes found

included rarefaction, sclerosis and exostosis.

Enlarged vascular channels (nutrient foramina) in the navicular bone have also been reported by Wilkinson (1952); Cawley (1960); Hickman (1964); Wintzer (1964); Colles and Hickman (1977); Rose et al. (1978) and others.

Cawley (1960) stated that the distal nutrient foramina were normally present in the navicular bone, but that in navicular disease, these enlarged and changed in shape to "mushroom or funnel shapes, and show lacunae".

Morgan (1972) maintained that a "saw toothed" appearance radiographically of the distal border of the bone was obviously pathological, but difficult to interpret correctly, whereas Colles and Hickman (1977) and Reid (1980) considered the presence of nutrient foramina in the distal border of the bone as physiological rather than pathological. Although often considered diagnostic of navicular disease, they maintained that the foramina were only of significance if they showed an alteration in their normal conical shape. Colles (1982) observed that in the normal horse there were up to seven nutrient foramina present in the distal border of the navicular bone, these were of a triangular shape and their height was $1\frac{1}{2}$ times their width at the base. He then described a further seven shapes of nutrient foramina which were found on radiographs from normal horses and those with navicular disease. Colles (1982a) considered that nutrient foramina occurring in the wings or proximal border of the navicular bone should be regarded as abnormal, even though they may be of a normal conical shape.

Dik, Nemeth and Merkens (1978) in contrast, considered the height of the nutrient foramina in the distal border of the bone as



a significant factor in the diagnosis of navicular disease, and disregarded their morphological change.

Attempts have been made by various workers to find objective criteria for the radiographic diagnosis of navicular disease based on these nutrient foramina. Rose et al. (1978) recorded the number of vascular channels, and measured the width and length of the channels, from the dorso-volar radiographs of navicular bones of normal horses and those with navicular disease. Although significant differences were found between the normal control group and the navicular disease group of horses, an overlap in values existed between the groups. For this reason they considered that this method was of no diagnostic value in the individual case.

Huskamp and Becker (1980) assessed navicular bones radiographically using quantitative criteria based upon the height as well as the change in shape of the nutrient foramina in the distal border. This system was applied to pre-purchase radiographs.

Other radiographic changes include areas of osteoporosis or rarefaction in the navicular bone. This lesion is considered by most workers (Pryer, 1934; Oxspring, 1935; Olsson, 1954; Hickman, 1964; Wintzer, 1964 and others) to be a significant finding occurring at a more advanced stage of the condition. On the other hand, osteoporotic areas can occasionally be found in the navicular bones of normal sound horses (Colles, 1979; Campbell, 1980). Colles (1979) reported that osteoporotic areas, which involved the tendinous surface of the bone and overlying fibrocartilage, could be demonstrated on a lateral radiograph.

Spur formation on the extremities of the proximal border of the navicular bone was considered to be diagnostic by both Pryer (1931a)

and Oxspring (1935), although as Pryer (1934) pointed out, these were not found in every case of navicular disease. Hickman (1964) reported that he had found spurs present in 10% of navicular disease cases and he considered small spurs to be of little significance, while Ackerman et al. (1977) noted that spurs were present in most of the navicular disease cases they examined. According to Numans and Vander Watering (1973) spur formation was not diagnostic, since it could be found in older normal horses. Colles (1982) classified spurs into three categories according to size. He concluded that spurs were more commonly found on the lateral side than on the medial side of the navicular bone, confirming the distribution noted by Morgan (1972). Colles also concluded that only large spurs, extending beyond the margins of the second phalanx, may be significant in navicular disease.

Osteophyte formation extending along the proximal border of the navicular bone has been reported as a radiographic finding in navicular disease by a number of workers (Oxspring, 1935; Hickman, 1964; Douglas and Williamson, 1970, Morgan, 1972 and others) while Colles (1982) also noted the presence of new bone growth along the distal border of the bone.

Chip fractures of the distal border of the navicular bone were reported to have occurred in 0.3% of navicular disease cases according to Van der Watering and Morgan (1975). In the cases where chip fractures were found, these were in addition to other radiographic changes of navicular disease.

Fracture of the navicular bone in a vertical plane has been considered to be a sequel to chronic navicular disease (Smythe, 1961; Vaughan, 1961; Hickman, 1964; Morgan, 1972; Adams, 1974), although it can occur following a trauma to the foot (Frecklington and Rose, 1981).

Colles (1982) considered fracture of the navicular bone to be a radiographic lesion indicative of navicular disease.

Increased density of bone or osteosclerosis, surrounding areas of rarefaction or around nutrient foramina, has been noted by a number of workers, (Jones, 1938; Wilkinson, 1952; Olsson, 1954; Cawley, 1960; and others), and is considered to occur in the later stages of the condition.

Colles (1982) maintained that in some horses a distinct notch was visible in the distal border of the navicular bone on a lateral radiograph, which, although it was found in normal horses, had a significantly higher incidence in navicular disease cases.

Oxspring (1935), Olsson (1954), Douglas and Williamson (1970), Adams (1974) and Colles (1982), all have stressed the importance of obtaining high quality radiographs of the navicular bone.

Colles (1982) concluded that;

No individual factor may be taken as certain evidence of clinical navicular disease and that even the presence of all of them need not be accompanied by clinical lameness. The more changes present, however, the more likely is clinical navicular disease to be present.

CHAPTER 3

PART I

RADIOGRAPHIC TECHNIQUE

INTRODUCTION

A radiograph has been defined as "a two-dimensional, flat-plane image of a three-dimensional subject" (Carlson, 1977), the purpose of the radiograph being as an aid to the diagnosis of disease (Oxspring, 1936).

Radiographic diagnosis involves both the location and identification of a lesion, and in order that the lesion can be accurately located a minimum of 2 views of the subject, preferably at 90° to each other, are required.

Various views of the navicular bone and various techniques for obtaining these views have been developed over the past 50 years. It was considered necessary in this study to examine some of these views and techniques in order to find the best method which combined technical ease with the production of high quality diagnostic radiographs.

Consistency in the end product was also required of the method, since it was intended that the navicular bones of a number of horses were to be radiographed at regular intervals over a period of time, and each set of radiographs would be compared with the previous set.

Douglas and Williamson (1963) considered the requirements of a diagnostic radiograph were that;

1. It should accurately portray the structures being examined with the minimum of distortion.
2. The structures under examination should be clearly discernible.
3. There should be no misleading artefacts.

As far as the last requirement is concerned, it has become the accepted practice before radiography of the navicular bone, to remove the shoe and clean and pare the foot in order to reduce the

possibility of artefacts.

This still leaves the shadows of the lateral and central clefts of the frog which would overly the navicular bone on the radiograph. It is generally considered that these clefts should be packed with a material of roughly the same radiographic density as the horn of the sole. However, some workers consider that these clefts should be left so that they may be identified clearly on the radiograph (Douglas and Williamson, 1970).

PACKING MATERIAL

MATERIALS AND METHODS

In this study, in keeping with the review of the literature, the term antero-posterior has been used instead of dorso-palmar, to describe a radiographic view made when the x-ray beam strikes the dorsal aspect of the limb and emerges on the palmar aspect.

Both front feet of 2 horses known to have bilateral navicular disease were prepared in the normal manner. Antero-posterior views were taken of the left fore foot, in each case using the upright pedal route with the hoof wall at an angle of 80° to the horizontal, and of the right fore foot, in each case using the high coronary route with the x-ray beam angled at 60° to the horizontal. Three antero-posterior views were taken of each foot with;

1. No packing material
2. Soft soap - as packing material
3. Play-doh - as packing material

Fast tungstate screens* and fast film⁺ were used with an 8:1 ratio

* Ilford Films, Ltd, London, England.

+ Fuji RX Film, Fujimex, Swindon, Wiltshire.

grid and a focus film distance of 90 centimetres (cm). The exposure factors used were, 78-84 Kilovolts (kV), 30 milliamps per second (mAS).

RESULTS

The quality of the films taken with no packing in the clefts of the frog were considered unacceptable as diagnostic films. Virtually no difference was found between the films taken using either soft soap or play-doh packing. From the point of view of use, the play-doh was more acceptable than the soft soap, which was difficult to retain in the foot.

DISCUSSION

Some workers believe that it is best not to pack the lateral and central clefts of the frog, so that these are identifiable on the radiograph, and not mistaken for pathological changes. In the films obtained using no packing, the full outline of the distal border of the navicular bone was not clear. Colles (1982) showed that soft soap was the packing material with the radiographic density nearest that of the horn of the sole, but in practice when the high kVs required for radiographing the navicular bone are used, little difference is noticed between the soft soap and play-doh.

CONCLUSIONS

The lateral and central clefts of the frog should be packed with a suitable material in order to obtain a clearer image of the navicular bone. Care must be taken in doing this to avoid the creation of artefacts which may resemble pathological changes in the image of the navicular bone. Either soft soap or play-doh were found to be suitable materials, but because of ease of use play-doh was the material of choice.

RADIOGRAPHIC TECHNIQUES

MATERIAL AND METHODS

Four horses were used in this work, 2 were sound control horses, and 2 were known cases of navicular disease. In each case both front shoes were removed, the feet cleaned and pared, and the lateral and central clefts of the frog packed with play-doh. All radiographs were taken using fast tungstate screens, fast film and a parallel 8:1 ratio grid (except where stated otherwise). The exposure factors ranged from 76kV-84kV and 30 mAS with a 90 cm focus-film-distance. All 4 horses had the navicular bones of both front feet radiographed using the following different views and techniques.

- (i) A latero-medial view - this was taken with the leg extended forward, the foot placed flat on a wooden block. Latero-medial views were taken with and without the use of a parallel 8:1 ratio grid.
- (ii) An antero-posterior view - using the upright pedal route with the front of the hoof wall angled at 90° to the horizontal. In order to achieve this positioning the foot was placed in a specially designed wooden block based on that of Tavernor and Vaughan (1962).
- (iii) An antero-posterior view - using the upright pedal route with the front of the hoof wall at an angle of 80° to the horizontal. The same wooden block as in (ii) was used for this purpose.
- (iv) An antero-posterior view - using the high coronary route with the x-ray beam angled at 60° to the horizontal. In order to achieve this positioning the horse's foot is placed on a reinforced cassette laid horizontally on the ground.

- (v) An antero-posterior view - using the high coronary route with the x-ray beam angled at 45° to the horizontal. The foot was placed on the cassette as in (iv) but in this case the angle of the x-ray beam was altered.
- (vi) A dorso-palmar view - this was taken using the method described by O'Brien et al. (1975). The horse was positioned so that it was standing on a reinforced cassette on the ground with the foot in its normal position or slightly caudal to its normal position. The x-ray beam was centred between the bulbs of the heels and angled at approximately 30° to the horizontal.

The 4 horses were destroyed, the navicular bones were dissected out, any gross pathological lesions recorded, and the bones were then radiographed intact. Several antero-posterior views were taken of each bone. The bones were placed flexor surface down on the cassette and then slightly angled, so that the anterior and posterior edges of the distal border were parallel. The radiographs were taken using high definition screens and fast film, a 90 cm focus-film-distance, and the exposure factors were 60-66 kV and 25-30 mAS.

Any radiographic changes present in the isolated navicular bone were then recorded and compared with these changes found in each of the different projections of the navicular bone.

RESULTS

Antero-posterior View

The radiographs of the four different antero-posterior views (ii-v) of the navicular bone of horse A42 are reproduced in FIGS 3.1-3.4. From these it can be seen that using:

- (ii) The upright pedal route with the hoof wall at 90° to the horizontal - there is only a slight foreshortening of the navicular bone image, and the proximal border of the bone is well defined, but the distal border of the navicular bone has not been projected above the level of the pedal joint. As a result of this the foramina in the distal border of the bone are not clearly visible.
- (iii) The upright pedal route with the front of the hoof wall angled at 80° to the horizontal - there is minimal distortion of the navicular bone image, and both the proximal and distal borders of the bone are clearly defined. The distal border of the navicular bone has been projected above the pedal joint and the nutrient foramina are distinct.
- (iv) The high coronary route with the x-ray beam angled at 60° to the horizontal - there is a slight lengthening of the navicular bone image using this route. As a result of this the proximal border is not as well defined as in (iii) but the distal border is clear, and projected above the level of the pedal joint, so that the nutrient foramina are evident.
- (v) The high coronary route with the x-ray beam angled at 45° to the horizontal - this resulted in marked distortion and magnification of the navicular bone image, and failure to project the distal border of the bone above the level of the pedal joint.

The results of the 4 antero-posterior views of the navicular bones in the other 3 horses were analagous with those above. The antero-posterior view of the navicular bone in situ, which most nearly equates to the radiograph of the isolated specimen, is that taken

using the upright pedal route with the front of the hoof wall angled at 80° to the horizontal. (FIGS 3.5 and 3.6)

Of the two routes, the upright pedal route was found to be much easier to carry out, in particular as regards aligning the foot, the film cassette, and x-ray beam relative to each other. With the high coronary route this alignment was more difficult to achieve, particularly with an unco-operative horse.

Latero-medial View

A latero-medial view was taken of each navicular bone, with and without the use of the grid. Better definition of the navicular bone image was obtained using a grid. The latero-medial view proved very useful for demonstrating 'spurs' or new bone growth on the proximal border of the navicular bone and for demonstrating radiolucent areas involving the flexor surface :

Difficulties were encountered in obtaining a true lateral view of the navicular bone, and unless this was achieved, the radiographs were extremely difficult to interpret. A better lateral view was obtained if the x-ray beam was centred on the navicular bone approximately $\frac{1}{2}$ inch below the coronary band and approximately 2 inches anterior to the heels, than when centred on the coronary band, and the x-ray cassette is positioned at 90° to the sagittal plane at the heels. (FIG 3.7)

Dorso-palmar View

Of all the 6 views taken of the navicular bone, this proved to be by far the most difficult to carry out. This was particularly so with an unco-operative or nervous horse, since the x-ray machine must be positioned in behind the limb being radiographed. This projection produced a clear view of the central part of the medullary

cavity, and the flexor surface of the bone. The nutrient foramina were evident in cross-section and radiolucent areas in the flexor surface of the bone were identified (FIG 3.8)

DISCUSSION

The first two requirements of a diagnostic radiograph were, that the radiographic image produced should be as accurate a portrayal as possible of the structure under investigation, and that the structure or areas being investigated should be clearly discernible.

In this study the subject under investigation is the navicular bone, which has inherent difficulties from the radiographic point of view, associated with its anatomical position within the hoof. The areas of importance in the navicular bone are the distal and proximal borders and the flexor surface. A minimum of 2 different radiographic views of the bone are required to show these areas.

The antero-posterior view which best fulfills the requirements is that obtained using the upright pedal route with the front of the hoof wall angled at 80° to the horizontal. This results in the least distortion of the navicular bone image. Work done by Campbell and Lee (1972) showed that this route resulted in less magnification of the navicular bone image than either of the high coronary routes. A clear view of the proximal border of the navicular bone was produced using the upright pedal route with the front of the hoof wall at 90° to the horizontal, but a degree of distortion of the navicular bone image occurred.

The antero-posterior view of the navicular bone, using the high coronary route with the x-ray beam angled at 45° to the horizontal, did not fulfill either of the requirements above, and the result was

considered to be unacceptable as a diagnostic radiograph.

Using the high coronary route with the x-ray beam angled at 60° to the horizontal gave clear views of the proximal and distal border of the bone, but there was slight distortion and magnification of the navicular bone image. From the diagnostic point of view there is not much difference between the upright pedal route with the hoof wall at 80° to the horizontal, and the high coronary route with the x-ray beam angled at 60° to the horizontal. The upright pedal method was found to be easier to carry out than the high coronary route. It was also found that the results were more consistent using this method, which is probably related to the technical ease rather than a feature of the route. However even using this route, it was found that when 2 antero-posterior views were taken of the same foot, the results were not always exactly the same. If the angle of the front of the hoof wall was increased or decreased in the upright pedal route, this resulted in distortion of the navicular bone image and of the proximal and distal borders.

Of the other two views, lateral and dorso-palmar, the lateral view was considered a better adjunct to the antero-posterior view than was the dorso-palmar view. The lateral view was technically easy, and allowed investigation of the proximal and distal borders and the flexor surface of the bone.

Although the dorso-palmar view gave a good skyline effect of the flexor surface, not as much information could be obtained from this view as the lateral view, and technically a lot of problems were associated with its use.

For the lateral view of the navicular bone to be of any diagnostic value it must be a true lateral. In order to demonstrate

finer details of the navicular bone on the lateral view, a parallel 8:1 ratio grid was used. This meant that there had to be careful alignment of the navicular bone, the film, and x-ray beam, but the end product showed far more detail than without the grid.

CONCLUSIONS

It was concluded from this work that, to obtain the best diagnostic radiographs of the navicular bone, a minimum of 2 views are required.

1. An antero-posterior view - using the upright pedal route with the front of the hoof wall angled at 80° to the horizontal.
2. A latero-medial view - with the x-ray beam centred on the navicular bone.

However, it is stressed that this is a minimum requirement. Since it was found that when 2 antero-posterior views were taken of the same navicular bone, using the same method, the results were not always identical, it is considered advantageous to take 2 antero-posterior views of the navicular bone, using the technique described above, as well as a latero-medial view.

CHAPTER 3

PART II

RADIOGRAPHIC FEATURES

INTRODUCTION

Considerable difference in opinion exists regarding the presence or absence of radiographic changes in the navicular bone, and their significance in navicular disease. Some workers believe that radiographic changes are always present in navicular disease cases (Rose et al., 1978; Colles, 1982), while others maintain that in many cases of navicular disease, no radiographic changes are present (Adams, 1974; Ackerman et al., 1977).

Few reports of the radiographic appearance of the navicular bones of sound horses have been made (Colles, 1982). It is therefore difficult to assess the significance of some of the radiographic changes found in navicular disease. It was considered important to examine radiographically the navicular bones of the control horses as well as those showing clinical signs of navicular disease.

MATERIALS AND METHODS

Following clinical examinations, the 265 horses used in this study were classified according to their histories and clinical features into Group A - control horses, Group B - horses with navicular disease, and Group C - horses with an undiagnosed foot lameness.

After this, radiographic examination of the navicular bones of both fore limbs, and, in some cases, the navicular bones of the hind limbs, was carried out.

Radiographic examination in each case involved removal of the shoe if present, careful cleaning and paring of the foot, and packing of the clefts of the frog with play-doh. Two antero-posterior views of the navicular bone were taken using the upright pedal route with

the hoof wall at 80° to the horizontal, the foot positioned in a wooden block and the x-ray beam centred on the coronary band. In addition, a latero-medial view of the navicular bone was taken with the leg extended forward, the foot positioned flat on a wooden block, and the x-ray beam centred on the navicular bone.

In the early stages of the work all the radiographs were taken using a Deans D-44 x-ray diagnostic machine*, fast tungstate screens, fast film, a parallel 8:1 ratio grid and a 90 cm focus-film-distance. The exposure factors used ranged from 78 to 84 kV, 25 to 30 mAS.

In the later stages of the work all the radiographs were taken using a Siemens Gigantos 1012 x-ray diagnostic machine+, Lanex rare earth screens#, fast film, a parallel 8:1 ratio grid, and a 90 cm focus-film-distance. The exposure factors used were 63-70 kV, and 20 mAS.

It is considered by some workers that the age of a horse may affect the radiographic appearance of the navicular bone. Numans and Van der Watering (1973) maintained that the development of nutrient foramina in the navicular bone was related to age, and that these were often absent in young horses up to 3 years old. Since in this study there were no horses less than 4 years old in the navicular disease group or in the NYD group, for the purpose of comparison, the radiographs of the horses less than 4 years old in the control group were excluded from this part of the study. This left 83 horses in the control group (GROUP A).

* A E Dean & Co Ltd, Croydon, England.

+ Siemens Ltd, Sunbury-on-Thames, England.

Kodak Ltd, Manchester, England.

Radiographic examination of the navicular bones of both front feet was carried out in 80 of the control horses, 119 of the navicular disease group, and 11 of the NYD group, and of one front foot, in 3 of the control horses, 32 of the navicular disease group, and 13 of the NYD group.

In addition to this, the same type of radiographic examination was made of the navicular bones of the hind limbs of a number of horses, 12 from the control group, 10 with no evidence of hind limb lameness from the navicular disease group, and 7 with clinical evidence of hind limb navicular disease from the navicular disease group.

The radiographic features of the navicular bone which were recorded in each case were:

- (i) The number of nutrient foramina visible in the distal border and the distal border of the wings.
- (ii) The number of these nutrient foramina which were conical in shape.
- (iii) The number of these nutrient foramina which were other than conical in shape.
- (iv) The presence of increased density in the distal border of the navicular bone and around the nutrient foramina.
- (v) The presence of nutrient foramina in the proximal border.
- (vi) Spurring on the medial and/or lateral extremities.
- (vii) Bone growth extending along the proximal border.
- (viii) Areas of radiolucency within the body.
- (ix) Chip fractures of the distal border, and the distal border of the wings.
- (x) Fracture extending from the proximal to distal borders.
- (xi) The presence of a distinct notch or groove in the distal

border on a latero-medial radiograph.

- (xii) Indentation of the flexor surface on a latero-medial radiograph.

The differences in number of distal nutrient foramina in the navicular bone was assessed by analysis of variance (TABLES 3.1 and 3.2). The differences between the groups of horses in the incidence of radiographic findings were tested for significance against chi-squared distribution. (TABLE 3.3).

RESULTS

The radiographic findings in the navicular bones of the front feet of the control horses (Group A), the horses with clinical evidence of navicular disease (Group B), and the horses with an undiagnosed foot lameness, are contained in APPENDICES 3.1, 3.2 and 3.3 respectively. The radiographic findings in the navicular bones of the hind feet of horses from groups A and B are contained in APPENDICES 3.4, 3.5 and 3.6.

NUTRIENT FORAMINA

The mean number of nutrient foramina detectable in the distal border and wings of the navicular bones of the front feet was calculated for each of the groups of horses. The mean number of nutrient foramina per navicular bone was 5.4 in the control horses, 6.5 in the NYD group, and 7.6 in the navicular disease group. There was a statistically significant difference between the mean number of nutrient foramina per navicular bone in each of the groups.

The number of these nutrient foramina in the distal border and wings of the navicular bone of the front feet which were conical in shape, and the number which were shaped other than conical, were

calculated. The mean number of conical nutrient foramina per navicular bone and the mean number of nutrient foramina shaped other than conical for the control group, NYD group, and navicular disease group are given in TABLE 3.1.

Significantly more conical nutrient foramina were therefore found in the navicular bones of the control horses than in the horses in the navicular disease group, and significantly more foramina, shaped other than conical, were found in the navicular bones of the navicular disease group than in the other 2 groups. In the navicular bones of the NYD group there were significantly more non-conical shaped nutrient foramina than in the navicular bones of the control group, but no significant difference was found in the number of conical shaped nutrient foramina between the 2 groups.

When the anatomical position of the nutrient foramina in the distal border of the navicular bone was examined, it was found that 8.3% of the foramina in the control group were in the wings of the navicular bone, and in the navicular disease group 15.6% of the foramina, a significantly higher percentage, were found in the wings of the navicular bone.

Nutrient foramina, evident in the proximal border of the navicular bone, were found in the navicular bones of the front feet in only 1 horse in the control group, in none of NYD group, and in 13 horses, a significantly higher number, in the navicular disease group, 6 bilaterally and 7 unilaterally.

Although only a small number of navicular bones of hind feet were radiographed, when the mean number of nutrient foramina per navicular bone was calculated, it was found that in those horses from group B with clinical evidence of navicular disease in the hind

limbs, there was a significantly higher number of foramina per bone than in the hind navicular bones of horses from the control group, and horses from the navicular disease group with no evidence of hind limb lameness (TABLE 3.2.).

It was also found that the horses with clinical evidence of navicular disease in the hind limbs had a significantly higher mean number of non-conical nutrient foramina than the hind navicular bones of the other horses.

No significant difference was found between the radiographs of the hind navicular bones of the control horses and the horses from the navicular disease group with no evidence of hind limb lameness, in the mean number of nutrient foramina per bone, the mean number of conical shaped foramina per bone, and the mean number of non-conical nutrient foramina per bone.

No difference was found in the distribution of nutrient foramina in the distal border of the hind navicular bones between the 3 groups of horses.

Nutrient foramina in the proximal border of the navicular bone were found only in the hind navicular bones of horses with evidence of hind limb lameness.

INCREASED RADIODENSITY

Increased density of the distal border of the navicular bone and around the nutrient foramina was found in 39(23.9%) of the navicular bones of the front feet in the control group, in 16(45.7%) of the navicular bones in the NYD group, and in 197(72.9%) of the navicular bones in the navicular disease group. There was a significant difference between each of the groups in the incidence of

increased density in the distal border of the navicular bones of the front feet. A larger percentage (80%) of the hind navicular bones of the horses with clinical evidence of navicular disease in the hind feet showed increased radiodensity in the distal border, but no significant difference was found between the groups in the incidence of increased radiodensity in the distal border of the hind navicular bones.

SPUR FORMATION

Spur formation on the medial and/or lateral extremities of the navicular bone was recorded in 33(20.2%) of the navicular bones in the control group. They were present in both front feet in 12 horses, and one front foot in 9 horses. In the NYD group, spurs were noted in 5(14.3%) of the 35 feet radiographed, 3 unilateral and 1 bilateral. Spurs were found in 68(25.1%) of the navicular bones in the navicular disease group. Both front feet were affected in 23 cases and one front foot was affected in 22 cases.

Spur formation was found more often on the lateral extremity than on the medial extremity, and this trend was present in all 3 groups of horses. Of the total number of spurs recorded, 64% were on the lateral extremity and 36% were on the medial extremity. No significant difference was found in the incidence of spur formation on navicular bones of the front feet between each of the 3 groups.

When the navicular bones of the hind feet were examined, no significant difference was found in the incidence of spur formation between the control group, the navicular disease group with no evidence of hind limb lameness, and the navicular disease group with clinical evidence of hind limb lameness. Also, no significant

difference was found between the fore and hind navicular bones in the occurrence of spurs. Although the numbers are small, no particular distribution of the spurs was noted in the hind navicular bones (47% lateral, 53% medial).

BONE GROWTH ON THE PROXIMAL BORDER

Bone growth on the proximal border at the insertion of the suspensory ligament of the navicular bone was found on the radiographs of 6(3.7%) of the navicular bones in the control group, in 57(21.1%) of the navicular bones in the navicular disease group, and in 4(11.4%) of the navicular bones in the NYD group. There was a significantly greater number of fore limb navicular bones with new bone growth on the proximal border in the navicular disease group.

From the radiographs of the hind navicular bones, new bone growth on the proximal border was found in only 1 horse unilaterally.

RADIOLUCENT AREAS

Areas of radiolucency within the body of navicular bone were recorded in only 1(0.6%) of the navicular bones in the control group, in none of the navicular bones in the NYD group, and in 37(13.7%), a significantly higher number, of navicular bones in the navicular disease group.

Areas of radiolucency were present in the navicular bones of the hind feet in 3 horses, 1 bilaterally and 2 unilaterally. All were from the navicular disease group with clinical evidence of hind limb lameness.

CHIP FRACTURES OF THE DISTAL BORDER

Chip fracture of the distal border of the navicular bone was

not found in any of the navicular bones in the control group, or in the NYD group, but was found in 4 bones from the navicular disease group. In each case it was unilateral, and only 1 fragment was visible. There was no radiographic evidence of a callus associated with these chip fractures. In the radiographs of the navicular bones of the hind feet, no chip fractures of the distal border were found.

FRACTURES EXTENDING FROM THE PROXIMAL TO DISTAL BORDER

Such fractures were not found in any of the navicular bones of the front feet in the control and NYD group of horses, but were present in 4 horses, unilaterally, from the navicular disease group.

In each case the fracture was in a vertical plane, just medial or lateral to the central ridge. There was no radiographic evidence of a callus associated with these fractures.

Complete fractures extending from the proximal to distal border were not found in any of the hind navicular bones radiographed.

A DISTINCT NOTCH IN THE DISTAL BORDER

A true lateral view of the navicular bone was required before this feature could be recorded. It was present in 46(28.2%) of the navicular bones in the control group, in 14(40%) of the navicular bones from horses in the NYD group, and in 129(47.7%) of the navicular bones from the horses with navicular disease. Of the hind navicular bones, 6(27.2%) in the control group, 6(30%) in the navicular disease group with no evidence of hind limb lameness, and 4(40%) in the navicular disease group with evidence of hind limb lameness, had a distinct notch in the distal border.

The incidence of a distinct notch or groove in the distal border of the fore navicular bones was significantly greater in the

navicular disease group and NYD groups than in the control group. No such difference was found in the navicular bones of the hind limbs.

INDENTATION OF THE FLEXOR SURFACE

Indentation on the flexor surface of the navicular bone was only evident on a true lateral view of the bone. This was found in 11(6.7%) of the navicular bones in the control group, 9 unilaterally and 1 bilaterally. In the NYD group, 2(5.7%) showed indentation of the flexor surface, and in the navicular disease group, 40(14.8%) of the navicular bones had an indentation of their flexor surface.

Indentation on the flexor surface of the navicular bones of the hind feet was noted in 2(9.1%) bones from the control group, 1(5%) from the navicular disease group with no evidence of hind limb lameness, and in 3(30%) of the navicular bones in the navicular disease group showing evidence of hind limb navicular disease.

Although indentation of the flexor surface of the fore navicular bone was found in the control horses and horses from the NYD group, a significantly higher incidence was found in the navicular bones of horses with clinical evidence of navicular disease. Of the navicular bones of the hind feet radiographed, the number showing indentation of the flexor surface is too small for statistical analysis, but it would appear that it may be a significant finding in hind limb navicular disease.

DISCUSSION

One of the radiographic features most under contention in the diagnosis of navicular disease is the significance of the distal foramina. Some workers consider the presence on radiographs of nutrient foramina in the distal border of the navicular bone, regardless of their number and morphology, is indicative of navicular disease (Wintzer, 1964; Morgan, 1972), while others maintain that nutrient foramina are normally present in the navicular bone and that a widening (Wilkinson, 1952) or a change in shape from conical, confirmed the presence of navicular disease (Colles and Hickman, 1977; Reid, 1980).

In this radiographic survey of the navicular bones of control horses and horses with clinical evidence of navicular disease, nutrient foramina were present in the distal border of the navicular bones of the control horses, as well as in those with navicular disease. The presence per se of nutrient foramina in the distal border of a navicular bone on a radiograph, should not be used as a criterion in establishing a diagnosis of navicular disease.

The number of nutrient foramina identified in the distal border of the navicular bone was significantly increased in the navicular disease cases. This finding is in agreement with that of Colles (1982). In the majority of navicular bones of the control group of horses, the number of distal nutrient foramina found ranged from 4 to 7, and in the majority of navicular bones of the navicular disease group of horses, ranged from 6 to 9, although in some bones only 4 nutrient foramina were present.

An increase in the number of distal nutrient foramina on radiographs, is not, on its own, diagnostic of navicular disease.

Conical shaped nutrient foramina were found in this study in the navicular bone of the control horses, and in those with navicular disease, but accounted for a significantly greater number of the foramina in the control group than in the navicular disease group. Of the total number of distal nutrient foramina in the navicular bones of the control horses, 79.4% were conical in shape and the remaining 20.6% were shaped other than conical, while in the navicular disease group, the distribution was quite different, 48.6% of the foramina were conical in shape and 51.4% were non-conical in shape. As a result of this finding, and the fact that conical shaped nutrient foramina were found in all the horses examined in this survey, conical shaped distal nutrient foramina can be considered as normal.

Reid (1980) and Colles (1982) have both stated that nutrient foramina visible along the distal border of the wings of the navicular bone should be considered as abnormal, regardless of their shape. In this study a significantly greater incidence of nutrient foramina in the wings of the navicular bone was found in navicular disease cases than in the control horses. This increase was not as a result of an increase in the number of non-conical shaped nutrient foramina in the wings, as might be expected from results shown in the last paragraph, but rather as a result of an increase in the number of normal conical nutrient foramina. The proportion, of conical to non-conical nutrient foramina in the wings of the navicular bones in the navicular disease group, is similar to that found in the distal border of the navicular bones in the control horses.

James, Kemler and Smallwood (1980) using corrosion casting and contrast xeroradiography, described a separate arterial blood supply to the distal border of the medial and lateral wings of the navicular

bone, which was not associated with the distal arterial supply which gives rise to the nutrient foramina along the distal border of the bone (Colles and Hickman, 1977). It is likely therefore that the nutrient foramina which are found in the wings of the navicular bone develop from this specific arterial supply, independently from the nutrient foramina in the distal border.

Colles (1982) noted in his radiographic survey of navicular bones that nutrient foramina visible in the proximal border of the navicular bone were found exclusively in navicular disease cases. The results of the present survey confirm the findings of Colles (1982) although nutrient foramina were evident in the proximal border of both front feet of one of the control horses.

It has been suggested by some workers that nutrient foramina become evident in the proximal border of the navicular bone in the later stages of navicular disease (Wilkinson, 1952), while Cawley (1960) considered that this feature was found in the early stages of navicular disease. The clinical details of the horses with nutrient foramina evident in the proximal border of the navicular bone, were examined, and no relationship between this radiographic feature and the duration or severity of lameness was found. The navicular bones with nutrient foramina evident in the proximal border had several other radiographic features present, such as, radiolucent areas in the body of the navicular bone (53%), Spur formation (47%), bone growth on the proximal border (37%), and indentation on the flexor surface of the navicular bone in 63%. It is likely that the proximal nutrient foramina are only present in established cases of navicular disease.

The importance of increased radiodensity of the distal border of the navicular bone and around the nutrient foramina, in navicular disease, is difficult to assess, because although it was a radiographic feature which had a significantly higher incidence in the navicular disease cases, it was also found in 23.9% of the navicular bones of the control horses. The presence of increased radiodensity of the distal border of the navicular bone is not in itself indicative of navicular disease, but together with other radiographic changes consistent with a diagnosis of navicular disease, provides additional useful confirmatory evidence.

Other radiographic features which were significant findings in navicular disease cases were, bone growth on the proximal border of the bone, radiolucent areas in the body of the bone, a distinct notch in the distal border and indentation of the flexor surface of the navicular bone.

Bone growth on the proximal border of the navicular bone was recorded separately from spur formation, although they are often considered together (Tavernor and Vaughan, 1962, Morgan, 1972). In this survey only 35% of the navicular bones with bone growth on the proximal border also had spur formation.

Unlike bone growth on the proximal border of the navicular bone, spur formation was not a significant finding in navicular disease cases.

Spurs have been considered by a number of workers as being an important radiographic feature of navicular disease (Pryer, 1931a; Oxspring, 1935; Ackerman et al., 1977). Colles (1982) classified spurs into 3 types according to their size, and he suggested that large spurs, which extend beyond the margin of the second phalanx, may be a significant finding in navicular disease cases. The spurs

found in this radiographic survey were examined and classified into 2 groups; small spurs which did not extend beyond the margin of the second phalanx and; large spurs which did extend beyond the margin of the second phalanx. (TABLE 3.4).

Although there is an apparently greater number of large spurs in the navicular disease group, when statistically analysed there was no significant difference between the 2 groups of horses. From this study it is concluded that spur formation is not an important radiographic feature of navicular disease, and spur formation should be differentiated from bone growth along the proximal border of the bone.

Areas of radiolucency within the body of the navicular bone, apart from 1 navicular bone in a horse from the control group, were only present in the navicular disease cases, and is therefore considered to be an important radiographic finding in the diagnosis of navicular disease. The area of radiolucency in some cases was not confined to the medulla of the navicular bone, but on a latero-medial radiograph, could be seen to extend and to involve the cortical bone of the flexor surface. This demonstrates the importance of taking more than one radiographic view of the navicular bone, in order to locate any radiographic features.

A distinct notch in the distal border of the navicular bone and indentation of its flexor surface were only evident on a true lateral view of the navicular bone. Such a view of the navicular bone can be difficult to obtain unless the x-ray beam is centred on the navicular bone, and even then, as Colles (1982) pointed out, ossification of the lateral cartilages is often present, and this can be superimposed across the flexor surface of the navicular bone leading

to difficulties in interpretation.

Despite these difficulties, indentation of the flexor surface of the navicular bone was noted in a significantly greater number of the horses in the navicular disease group, and therefore should be regarded as a useful feature, together with other radiographic changes, in confirming a diagnosis of navicular disease.

A distinct notch in the distal border of the navicular bone, although it was present in quite a number of the navicular bones of the control horses (28%), had a significantly higher incidence in the navicular bones of the navicular disease group. The high incidence of this radiographic feature in navicular disease cases may be associated with the increased number of nutrient foramina in the distal border of the bone in navicular disease cases.

Two radiographic features which were found exclusively in the navicular disease group were, chip fracture of the distal border of the navicular bone, and complete fracture of the navicular bone extending from proximal to distal borders. Chip fracture of the distal border of the navicular bone is a difficult feature to detect on routine radiographic examination, and more than one antero-posterior view of the bone is required. In each case only 1 fracture fragment was present and only one foot affected. No evidence of a callus or other bone reaction was found. No relationship between the chip fractures and other radiographic findings of navicular disease could be established. Van der Watering and Morgan (1975) suggested that these chip fractures were avulsion type fractures rather than pathological fractures. Although only a small number of chip fractures were found, the results of this present survey are in accord with those of Van der Watering and Morgan (1975).

Fracture of the navicular bone was found in 4 cases of navicular disease. In each case several antero-posterior views of the navicular bone were taken with the front of the hoof wall at slightly different angles between 90° - 80° to the horizontal, and the foot re-packed on each occasion. This process was carried out because of the danger of artefacts, associated with the preparation of the foot, resembling fracture lines in the navicular bone. In each case the fracture was complete, vertical, extended from the proximal to distal borders and was positioned medial or lateral to the central ridge. This is the most common type of navicular bone fracture (Vaughan, 1961). All 4 cases had other radiographic changes of navicular disease.

Over the $3\frac{1}{2}$ year period of this study, 10 cases of fractured navicular bone in a fore foot have been recorded, and of the 10 cases only 4 had case histories, clinical signs, and radiographic features of navicular disease. It is evident from these findings that fracture of the navicular bone can occur as a sequel to navicular disease, but need not necessarily be associated with navicular disease.

The NYD group of horses has also been included in this comparative radiographic survey. All of these horses had a fore foot lameness of unknown aetiology, which fulfilled some of the clinical criteria for navicular disease, and it was considered important to compare the radiographic features of their navicular bones with those of the control and navicular disease groups.

In this NYD group, the incidence of some radiographic features fell between those of the control group and the navicular disease group. The NYD group had significantly more distal nutrient foramina per bone than the control group, but the navicular disease group had significantly more per bone than the NYD group. This was also true

of the number of non-conical shaped distal nutrient foramina per bone in the NYD group, although the number of normal conical nutrient foramina per bone was similar to that of the control group. The other radiographic findings, whose incidence was between that of the control horses and the horses with navicular disease, were increased radiodensity of the distal border and around the nutrient foramina, and the presence of a distinct notch in the distal border of the bone.

The radiographic features which were absent from the NYD group were, nutrient foramina in the proximal border of the bone, radiolucent areas within the body of the bone, chip fracture of the distal border, and fracture of the navicular bone. It is concluded from these findings that the NYD group of horses do not have radiographic features consistent with navicular disease, but, that some of the radiographic features demonstrated differ significantly from those of the control horses. It is possible that:

- (i) Some of these horses in the NYD group have a low grade, non-progressive or only slowly progressive form of navicular disease, which gives rise to only some of the clinical manifestations and some of the radiographic features of the typical navicular disease case.

or that:

- (ii) As a result of disease processes elsewhere in the foot, there is a change in the vascularisation of the navicular bone, producing radiographic changes in the navicular bone.

The radiographic findings in the NYD group of horses are somewhat similar to those of the "chronic fore foot lameness" group of horses examined by Colles (1982). In his chronic lameness group, a diagnosis of both navicular disease and chronic laminitis had been

definitely ruled out.

A small number of hind navicular bones were examined radiographically in this study, and where possible the findings were subjected to statistical analysis. The hind navicular bones of 3 groups of horses were examined, horses from the control group, horses from the navicular disease group that had clinical evidence of hind limb navicular disease as well as forelimb navicular disease, and horses from the navicular disease group with no evidence of hind limb lameness. This latter group was included in order to establish whether, as a result of fore limb navicular disease in these horses, there was any difference in the radiographic features of the hind navicular bones.

The radiographic findings in the hind navicular bones are similar to those of the fore navicular bones, except that, bone growth on the proximal border, chip fracture of the distal border, and complete fracture of the navicular bone was not present in any of the hind limbs examined. No difference was found between the radiographic features of the hind navicular bones of the control horses and the hind navicular bones of the horses from the navicular disease group with no hind limb lameness. It is concluded from this that the navicular bones of the hind feet in which there was no clinical evidence of navicular disease, are normal, despite there being navicular disease present in the fore feet.

The results of radiographic examination of the navicular bones in the horses with clinical evidence of navicular disease in the hind feet are similar to those of Valdez et al. (1978) who reported the clinical findings and radiographic changes in 6 horses with navicular disease of the hind limbs.

Some workers consider that in many cases of navicular disease, radiographic changes in the navicular bone are not present. (Carlson, 1967; Adams, 1974; Ackerman et al., 1977 and others), while Rose et al. (1978) and Colles (1982) maintain that radiographic changes are always present in the navicular bones in navicular disease cases. In this study radiographic changes were present in the navicular bones of all the limbs in which there was clinical evidence of navicular disease. The results therefore of this present radiographic survey confirm the findings of Rose et al. (1978) and Colles (1982).

CONCLUSIONS

It is concluded from the results of routine radiographic examination of 83 control horses, 151 horses with clinical evidence of navicular disease, and 24 horses with an undiagnosed foot lameness that:

- (i) Radiographic changes were present in all cases of navicular disease examined.
- (ii) No one radiographic feature was diagnostic in itself of navicular disease, and it is therefore important to consider all the radiographic features present before confirming a diagnosis of navicular disease.
- (iii) The radiographic features which are considered important in confirming a diagnosis of navicular disease are:
 - (a) An increase in the number of nutrient foramina in the distal border of the navicular bone.
 - (b) A change in shape of the nutrient foramina in the distal border of the bone from a normal conical shape.
 - (c) The presence of nutrient foramina in the proximal border of the navicular bone.
 - (d) Radiolucent areas in the body of the navicular bone.
 - (e) Indentation on the flexor surface of the navicular bone.
- (iv) As these changes, and the other radiographic changes of navicular disease, can also be found in horses that are not lame, it is stressed that radiography should not be used to diagnose navicular disease, but to confirm a clinical diagnosis.

CHAPTER 3

PART III

RADIOGRAPHIC ASSESSMENT

THE IDENTIFICATION AND CLASSIFICATION OF DIFFERENT TYPES OF DISTAL NUTRIENT FORAMINA IN THE NAVICULAR BONE

INTRODUCTION

Colles and Hickman (1977) described the blood supply to the navicular bone and its development. There are 2 main sources, a proximal and distal arterial supply. Arteries along the distal border of the bone gradually penetrate the bone, and, by the time the horse is 2 or 3 years old, form cones of anastomosing vessels, which bring about the development of the nutrient foramina visible on radiographs of the navicular bone. These distal nutrient foramina are considered to be normally conical or triangular in shape (Colles and Hickman, 1977; Reid, 1980; Rose, 1981).

The results shown in Part II indicate that the number and morphology of the distal nutrient foramina are an important feature in the radiographic diagnosis of navicular disease.

From radiographic examination of the navicular bones of 258 horses used in this study, it has been shown that there is a significant increase in the number of distal nutrient foramina in the navicular bones in navicular disease cases. There is a decrease in the number of normal conical shaped nutrient foramina, while there is an increase in the number of abnormally shaped foramina.

In view of these findings a further, more detailed study of the nutrient foramina was undertaken. This study was directed towards identifying and classifying the foramina according to morphology and position..

MATERIALS AND METHODS

The antero-posterior radiographs of the navicular bones of the 83 control horses (GROUP A), the 151 horses with navicular disease

(Group B), and the 24 horses with an undiagnosed foot lameness (Group C), were re-examined, the different shapes of nutrient foramina in the distal border and distal border of the wings of the navicular bones, were recorded, and the number analysed. To assess differences in incidence of each type of foramen, significance tests were based on chi-squared. As far as possible this examination was carried out without the examiner knowing from which groups the radiographs were taken.

In addition, it was possible to examine radiographs of the navicular bones of a small number of untreated cases of navicular disease, and of 5 control horses, taken at intervals over a period of up to 2 years.

This allowed monitoring of any alteration in numbers and shape of the distal nutrient foramina in this period.

RESULTS

From re-examination of the antero-posterior radiographs of the navicular bones, 7 basic types of distal nutrient foramina were identified. The numbers of each type of nutrient foramen occurring in each of the groups of horses, and the percentage of incidence of each type out of the total number of distal nutrient foramina in each group, has been calculated (TABLE 3.5). The results of statistical analysis are also contained in TABLE 3.5.

The normal conical shaped nutrient foramina present in the distal border of the navicular bone, and the normal conical shaped nutrient foramina present in the distal border of the wings of the navicular bone, were separated into different categories, type A and type B respectively.

- Type A - This is a conical/triangular shaped nutrient foramen, wider at its base than at its top. A highly significant incidence of this type of foramen was found in the navicular bones of the control group. In the NYD group, the incidence of conical shaped nutrient foramina in the distal border of the navicular bone lay between that of the control group and the navicular disease group, but also had significantly more conical nutrient foramina than the navicular disease group.
- Type B - This is a similarly shaped nutrient foramen to type A, but in this case it is found in the distal border of the wings of the navicular bone. A significantly higher incidence of this type of nutrient foramen was found in the navicular disease group. No difference in incidence of type B foramina was found between the control and NYD groups.
- Type C - Type C nutrient foramen is the same width throughout its length and is rounded at the top. It had a significantly higher incidence in the navicular disease group than in the control group, but no significant difference was found between the navicular disease group and the NYD group.
- Type D - Type D foramen is slightly wider at its top than at its base, and the top is rounded. It had a significantly higher incidence in the navicular disease group than in the control group. The numbers of this type of foramen found in the NYD group were too small for statistical analysis.
- Type E - This type of foramen, which is referred to as a 'goblet' shape, has a definite neck or stalk, and a widened circular top. It was found in significantly greater numbers in the navicular disease group than in the control or NYD groups.

Type F - Type F foramen is a branched foramen which is best described as a 'staghorn' shaped foramen. It has a neck or stalk and 2 distinct branches, which are rounded at their tops. This type of foramen is not common, and the numbers in each group were too small for statistical analysis. Nevertheless the numbers of type F foramina found in the different groups suggests a more frequent occurrence in the navicular bones of the navicular disease group.

Type G - Type G foramen is a variation of the 'staghorn' type of foramen. It has a definite neck or stalk, and two branches, which end in widened circular tops. Only 3 of this type of foramen were found, and these were exclusively in the navicular disease group.

Type H - Type H nutrient foramen has 2 or more necks or stalks which join onto a large circular or oval area of radiolucency, and were found exclusively in the navicular disease group.

Examples of some of these types of nutrient foramina are shown in FIGS 3.9 - 3.14).

Alterations were recorded in the shape and number of the distal nutrient foramina, from radiographic examination at intervals, of the navicular bones of a number of untreated navicular disease cases, over a period of up to 2 years (FIG 3.15).

The changes in shape of nutrient foramina recorded were:

- (i) a change from type A to types C or D.
- (ii) a change from type C to type D or E.
- (iii) a change from type E to type H.

An increase in the number of nutrient foramina present in the distal border of the bone was recorded in some cases. This increase

in number was as a result of the addition of type A and type B nutrient foramina.

An increase in number, or change in shape, of the distal nutrient foramina, did not occur in all horses for which serial radiographs were available, nor did the changes noted affect all of the foramina in an individual navicular bone. The untreated cases of navicular disease showed either no change in their degree of lameness, or had become progressively worse over the period of radiographic examination. Examination of the serial radiographs of the navicular bones of 5 control horses showed no increase in the number, or change in shape, of the distal nutrient foramina.

DISCUSSION

A change in shape of the distal nutrient foramina has been described as a radiographic feature of navicular disease by a number of workers. Wilkinson (1952) described a widening of the foramina in the distal border of the navicular bone in navicular disease, while Cawley (1960) maintained that the foramina enlarged into an abnormal 'mushroom' or 'funnel' shape. According to Reid (1980) a change in shape of the distal nutrient foramina, from a normal cone shape to a 'lollipop' shape, was indicative of navicular disease, and Rose (1981) described the abnormal nutrient foramina as being an 'inverted flask' shape. These authors described only foramina of the conical type, which they considered normal, and the mushroom or lollipop type, which they considered abnormal. This latter shape of nutrient foramina corresponds to the type E, goblet foramen, and possibly the type D foramen, described in this study. However, these types of distal nutrient foramina were not the only

non-conical shaped nutrient foramina to be found in the distal border of the navicular bone. Colles (1982), from his radiographic survey of navicular bones, described 8 different shapes of nutrient foramina, found in the distal border of navicular bones of control horses and those with navicular disease.

In this present radiographic survey, 7 different shapes of nutrient foramina were recorded, which are similar to those of Colles (1982). The normal conical shaped nutrient foramina were classified into 2 types according to anatomical position, type A being found in the distal border of the bone, and type B being found in the distal border of the wings of the bone. The conical nutrient foramina were ~~classified~~ in this way because the results in Part II of this work indicated that the presence of nutrient foramina in the wings of the navicular bone was a significant finding in navicular disease cases.

Nutrient foramina of types B, C, D and E, were all found in significantly greater numbers in the navicular bones of the navicular disease cases, and types G and H were found exclusively in the navicular disease cases. From these findings it is considered that all of the distal nutrient foramina, shaped other than conical, can be regarded as abnormal.

Some of these abnormal types of nutrient foramina were found in the navicular bones of the control horses. It is considered, therefore, that the presence of abnormal shaped nutrient foramina in the distal border of a navicular bone is not in itself diagnostic of navicular disease, but from the results of Part II of this work, it is suggested that the more nutrient foramina of abnormal shape that are present, the more likely is there to be clinical evidence of

navicular disease. Only type G and H foramina were found exclusively in navicular disease cases, and so, their presence in the distal border of a navicular bone should be regarded as an important finding in confirming a diagnosis of navicular disease.

Significantly more distal nutrient foramina per navicular bone have been found in the NYD group, than in the control group, but only type C had a significantly higher incidence in the navicular bones of the NYD group as compared to the control group. The incidence of the other abnormal shaped distal nutrient foramina in the NYD group was similar to that of the control group.

Radiographic examination will only demonstrate the radiographic changes present at the time of examination, and will give no indication of the development or progress of any changes. In this study it was possible to carry out serial radiographic examinations of the navicular bone of a number of control horses and horses with navicular disease. From these examinations, changes in the number and degree of the distal nutrient foramina were detected in some of the horses with evidence of navicular disease, but no such changes were found in the navicular bones of the control horses examined.

The change in shape of the distal nutrient foramina did not appear to be a random change, but rather, followed a distinct pattern. Change from a conical type A foramen to types C or D have been recorded, while the shape of other foramina has changed from types C and D to E, the goblet shaped foramen. Another change found was from type E to type H foramina. This was brought about by the enlargement and joining of two or more type E foramina to form one large radiolucent area.

These findings suggest that the different shaped distal

nutrient foramina recorded in this study are each part of a progressive series of changes, although these changes need not occur in all horses, or to all of the foramina in an individual navicular bone. The suggested progressive series of changes in the shape of the distal nutrient foramina is from a conical nutrient foramen type A or B to type C, from this to type D, E and eventually H, and although not recorded in the serial radiographs in this study, it is likely that type G foramina develop from type F foramina.

The rate at which the shape of the nutrient foramina changed varied markedly between horses. For example, the time taken for the development of a type H foramen from 2 type E foramina varied from 4 months in one horse to 18 months in another. It was impossible to determine the point at which one type of foramen becomes another type of foramen.

Changes in the shape of distal nutrient foramina, from type A, through the suggested progression to type H, were not recorded in any of the cases of navicular disease subject to serial radiographic examination. It is possible that the complete series of progressive changes in shape was not found, because the period of time over which the serial radiographs were taken was not long enough.

CONCLUSIONS

It is concluded from the identification and classification of the different types of distal nutrient foramina evident in navicular bones that:

- (i) Nutrient foramina, shaped other than conical, can be considered as abnormal.
- (ii) The presence of abnormal shaped distal nutrient foramina in a

navicular bone is not diagnostic of navicular disease, but the greater the number of abnormal nutrient foramina present in a navicular bone, the more likely are clinical signs of navicular disease to be present.

- (iii) No particular type of abnormal shaped nutrient foramen is diagnostic of navicular disease, since most types can be found in the navicular bones of control horses. However the presence of type G and/or H foramina in a navicular bone should be considered an important finding in confirming a diagnosis of navicular disease.

Examination of serial radiographs of the navicular bones of untreated navicular disease cases and control horses, led to the following conclusions:

- (i) The distal nutrient foramina may increase in number and/or show progressive alteration in their shape.
- (ii) The alteration in shape of the distal nutrient foramina does not appear to be a random change, and the different types of abnormal shaped nutrient foramina found are part of a series of progressive changes.
- (iii) Progressive change in the shape of the distal nutrient foramina of the navicular bone does not occur in all cases of navicular disease, and in those cases which do show changes, not all the distal nutrient foramina will be affected.

These findings suggested that it would be useful to develop a method of assessing the changes in shape of the distal foramina in the navicular bone.

QUANTITATIVE METHOD OF ASSESSING DISTAL NUTRIENT FORAMINA FROM RADIOGRAPHS OF THE NAVICULAR BONE

INTRODUCTION

The results of the radiographic survey in this study showed that the navicular bones of all the horses examined had nutrient foramina present in their distal borders. It has been established that conical shaped distal nutrient foramina are normal, and that nutrient foramina shaped other than conical, are abnormal. When the abnormal distal nutrient foramina were looked at in more detail, 6 different shapes of abnormal nutrient foramina were found. These 6 abnormal shaped nutrient foramina, and the normal conical shaped nutrient foramina, were present in different combinations in each navicular bone, and it has been shown that the shape of the nutrient foramina may change over a period of time in navicular disease cases.

An objective method of assessing the radiographs of navicular bones was desirable in order to compare:

- (i) The radiographs of the navicular bones of different horses.
- (ii) Serial radiographs of the navicular bones of the same horse.
- (iii) The radiographic findings with the clinical findings.
- (iv) The radiographic findings with the pathological findings.

The criteria used in establishing this objective method of assessment were, the number, shape, and anatomical position of the distal nutrient foramina.

MATERIALS AND METHODS

A scoring system was devised, whereby each nutrient foramen identified in the distal border of a navicular bone, on an antero-posterior radiograph, was given a score, from 1 to 12, which was dependent upon its shape, and/or its anatomical position. Scores

were given to nutrient foramina based on the series of progressive changes demonstrated in the previous section. The scores for each nutrient foramen present in the distal border of a navicular bone were then added together to give a total score for that particular navicular bone.

A minimum of 2 antero-posterior radiographs of a navicular bone were required before the bone was assessed by this method. The average score was then calculated for each navicular bone.

Scoring system for distal nutrient foramina in the navicular bone

The score given to each type of nutrient foramen is shown in FIG 3.16.

Type A - A normal conical shaped nutrient foramen, found in the distal border of the navicular bone, was given a score of 1.

Type B - A normal conical shaped nutrient foramen, found in the distal border of the wings of the navicular bone, was give a score of 2.

Type C - A type C nutrient foramen, whether it was present in the distal border, or in the distal border of the wings of the navicular bone, was given a score of 3.

Type D - A type D nutrient foramen, whether present in the distal border or wings of the navicular bone, was given a score of 4.

Type E - This goblet type of nutrient foramen was given a score of 5, whether it was present in the distal border or in the wings of the navicular bone.

Type F - A type F nutrient foramen, whether in the distal border or wings of the navicular bone, when present was given a score of 6, since it was considered to be equivalent to 2 type C

foramina.

Type G - This type of nutrient foramen, which was considered to be equivalent to 2 type E (goblet) foramina, was given a score of 10.

Type H - Those type H foramina, which consisted of a radiolucent area with 2 stalks, were given a score of 12, and with 3 stalks a score of 18.

This scoring system was then applied to the radiographs of the navicular bones of the 258 horses examined in this study. As previously, the radiographs were examined, and their scores assessed, without the examiner knowing from which group of horses the radiographs had been taken. An example of a radiograph of a navicular bone and the method of calculating its score is shown in FIG 3.17.

The navicular score for each horse was calculated, in the control horses and the horses with bilateral fore limb lameness, by taking the average score of both fore feet, and in the horses with unilateral fore limb lameness the score of the affected fore foot was taken as the navicular score for that horse.

All of the horses examined radiographically were then classified into one of 7 groups according to their navicular score.

This scoring system was similarly applied to the radiographs of the hind navicular bones.

The difference between the groups of horses, in the calculated score for each navicular bone, was assessed by analysis of variance.

RESULTS

The results of applying this scoring system to the radiographs of the fore navicular bones of 83 control horses, 151 horses with

navicular disease and 24 horses in the NYD group, are contained in Appendices 3.7, 3.8 and 3.9 respectively, and results of the hind navicular bones in Appendices 3.10, 3.11, and 3.12.

In the control group of horses the navicular scores per bone ranged from 3 to 15, with the mean navicular score per bone being 8.2. In the navicular disease group the navicular score per bone ranged from 8 to 46, with the mean navicular score per bone being 18.6. In the NYD group the navicular score per bone ranged from 4 to 18, and the mean navicular score per bone was 12.1 (TABLE 3.6)

A highly significant difference was found between the mean navicular score per bone of each of the 3 groups of horses (TABLE 3.6)

The mean navicular score per bone in the hind navicular bones, was, 6.9 in the control horses, 7.5 in the navicular disease group with no evidence of hind limb lameness, and 18.4 in the navicular disease group with evidence of hind limb lameness (TABLE 3.7). The mean navicular score per navicular bone was significantly higher in the navicular disease group with evidence of hind limb navicular disease than in the other two groups (TABLE 3.7).

The navicular score for each horse was then calculated and the horses classified into one of 7 groups according to their navicular score (TABLE 3.8).

All of the control horses were distributed between the first 4 groups, the majority of them being in the 5-9 score group. The horses in the NYD group were also distributed between the first 4 groups, but the majority of this group were in the 10-14 score group.

In the navicular disease group all horses had a navicular score greater than 10 and the majority of them were in the 15-19, 20-24 score groups (FIG 3.18).

The calculated navicular score for each of the horses in the navicular disease and NYD groups were compared to the degree of lameness in each case. A significant positive correlation was found between the navicular score and the degree of lameness in the navicular disease group (FIG 3.19), but no such correlation was found in the NYD group (FIG 3.20).

DISCUSSION

The diagnosis of navicular disease is based upon a series of subjective processes, which include the clinical examination and the radiographic examination.

Rose et al. (1978) using the objective criteria of measuring the width, length, and number of nutrient foramina on a dorso-palmar view of the navicular bone, compared the radiographs of a number of control horses, and horses with navicular disease. He found that although there was a significant difference between the measurements of the 2 groups, which was an aid to the diagnosis of navicular disease, these criteria were of little value in the individual case.

Huskamp and Backer (1980) described a scoring system for the nutrient foramina in the distal border of the navicular bone. This scoring system was based on the length and the shape of the nutrient foramina, and was used in confirming a diagnosis of navicular disease as well as in pre-purchase radiographic examinations of the navicular bone.

In this study it was decided that a standard method of assessing radiographs of the navicular bones of both control horses and horses with navicular disease was required, to allow comparison of, the radiographs of different horses, serial radiographs of the navicular bones of the same horse, the radiographic findings, clinical findings and pathological findings.

This method of assessment was based entirely upon the distal nutrient foramina of the navicular bone for a number of reasons.

- (1) Nutrient foramina were present in the distal border of the navicular bones of all the horses examined radiographically in this study.

- (ii) Significant differences have been shown in the number, anatomical position, and proportion of normal to abnormal shaped distal nutrient foramina, in the navicular bones of control horses and horses with navicular diseases.
- (iii) These changes in the distal nutrient foramina were present in all cases of navicular disease examined, whereas other radiographic features of navicular disease were not present in all cases, and some were only present in advanced cases of navicular disease. It is likely that different stages of navicular disease are represented in the 151 cases used in this study. Since changes in the distal nutrient foramina were present in all of them, then using these changes as criteria for assessing radiographs of the navicular bone may prove valuable in the diagnosis of early cases of navicular disease, which do not show other radiographic changes.
- (iv) The number of distal nutrient foramina and their shape and position are relatively easy to detect, providing the antero-posterior radiographs of the navicular bone are of the required standard.

The navicular scoring system devised takes into account the number, shape and anatomical position of the distal nutrient foramina on radiographs of the navicular bone. Dik et al. (1978) suggested a scheme for the radiographic diagnosis of navicular disease which takes into account the number and height of the distal nutrient foramina, but disregards their change in shape.

In this present study, no consideration is given to the height of the nutrient foramina, since considerable variation in height was found between the foramina in control horses, and Colles and Hickman

(1977) showed that the height of the nutrient foramina in the distal border of the navicular bone was related to the amount and frequency of the work performed by a horse. The results of measuring the height of the nutrient foramina varied considerably with only slight alteration in radiographic technique. This method of assessment is not entirely objective in nature, since a degree of subjectivity is employed in determining the shape of the distal nutrient foramina.

Significant differences were found between the mean navicular score per bone of each of the groups of horses. The results obtained for the NYD group lay between that of the control and navicular disease groups, although it had been shown in an earlier part of this work that only the incidence of type C foramina was greater in the NYD group than in the control group.

When the control, navicular disease, and NYD groups of horses were classified according to their navicular scores, a considerable overlap of the different groups of horses was found. This overlap was between the navicular scores 10 and 19.

This study did not allow a long term investigation of control horses in order to establish whether a sound horse, whose navicular score lay between 10 and 19, is more likely to develop navicular disease than any other sound horse.

These findings emphasize the danger of using a scoring system such as this one to determine whether a horse has or has not navicular disease. It is stressed again that radiography on its own should not be used to diagnose navicular disease, but to confirm a clinical diagnosis.

The navicular scoring system is considered very useful for comparing radiographs of the navicular bones of different horses,

and for recording serial radiographs of navicular bones of the same horse.

When the calculated navicular score of each horse in the navicular disease group was compared to their degree of lameness a positive correlation was found. Despite this correlation, wide variations were found between the navicular score and the degree of lameness, and it is concluded that the navicular scoring system should not be used to determine the degree of clinical involvement.

CONCLUSIONS

It is concluded that the navicular scoring system provides a useful method of assessing and comparing antero-posterior radiographs of the navicular bone, particularly in early cases of navicular disease.

However, the diagnosis of navicular disease should not be based upon the scoring system on its own, or on any particular value of navicular score, but rather the scoring system should be used as an aid in confirming a diagnosis of navicular disease.

It is emphasised that the value of the navicular scoring system is dependant on the quality of the radiographs taken. A minimum of 2 antero-posterior radiographs of each navicular bone are required, and the average score is calculated from these.

CHAPTER 4

PATHOLOGICAL FEATURES OF NAVICULAR DISEASE

It ain't the 'unting as 'urts 'un, it's the
'ammer, 'ammer, 'ammer along the 'ard 'igh road.

Punch Vol. 30 1856

REVIEW OF THE LITERATURE

The pathogenesis of navicular disease has always been a highly controversial area. It was believed by Turner (1829), Moorcroft (1830), Dick (cited by Smith, 1886) and Westhues (1938) that the disease originated in the deep flexor tendon, and from there disease spread to affect the navicular bursa, the fibrocartilage of the navicular bone and eventually the navicular bone itself. On the other hand Smith (1886) and Fitzwygram (1894) considered that the primary disease was an inflammation of the navicular bone, and other changes in the fibrocartilage and deep flexor tendon were subsequent to this. Percivall in 1873 and later, Adams in 1974 and Baum (1974) maintained that navicular disease began as an inflammation of the navicular bursa which, then, led to erosive and degenerative changes in the fibrocartilage of the navicular bone and in the deep flexor tendon. Colles (1982) defined navicular disease pathologically as any condition affecting the navicular bone and surrounding structures, which he further classified into four categories.

It was suggested by Hickman (1964) that navicular disease, "is not a specific entity, but rather the clinical symptoms observed are the manifestations of pain in the navicular bone due to a variety of distinct and separate pathological changes affecting it and its fibrocartilage". Most authors have agreed that navicular disease never affects the articular surface of the navicular bone (Turner, 1829; Reeks, 1925; Hickman, 1964) while Adams (1974) considered that the articular surface of the navicular bone was occasionally affected, leading to an arthritis of the coffin joint.

GROSS PATHOLOGICAL CHANGES

The earliest detailed account of the post mortem findings in navicular disease cases was by Smith in 1886. He described the pathological changes of the synovial membrane and synovia, the deep flexor tendon, the fibrocartilage surface of the navicular bone and the navicular bone itself. Unlike Turner (1829) and Percivall (1873), Smith (1886) could find no marked inflammatory change in the synovial membrane of the navicular bone.

He reported that in 99% of navicular disease cases there were patches of brown staining on the area of the deep flexor tendon in contact with the navicular bone, with varying degrees of erosion of tendon fibres in an upward direction. He noted similar patches of brown staining on the fibrocartilage surface of the navicular bone. He considered that this brown staining was probably derived from minute capillary haemorrhages in the eroded tendon. Oxspring (1935) agreed with these findings. This yellow-brown discolouration was observed by Wilkinson (1953) on both the fibrocartilage surface of the navicular bone and the deep flexor tendon, and following investigations into the nature of the pigment he decided that it was of fatty origin; "This discolouration seems to occur mainly in the tendons and fibrocartilage of old horses and it is probable that it is of a similar nature to the lipochrome pigment which occurs in the muscle fibres of the heart in the old human subject". He therefore concluded that the discolouration was unlikely to be of any significance in navicular disease. Colles (1982) considered from post mortem examination of navicular bones from normal horses and those with navicular disease that yellow discolouration and erosion of the fibrocartilage surface of the navicular bone were "normal wear lesions"

and not early changes of navicular disease.

Other changes reported by Smith (1886) were, calcareous deposits in the fibrocartilage of the navicular bone, ulceration of the fibrocartilage to varying degrees, hypertrophy, discolouration and caries of the navicular bone itself.

Earlier workers (Moorcroft, 1830; Brauell, 1846) reported what would appear to have been these calcareous deposits noted by Smith (1886). Smith (1886) observed that "if calcareous deposits are exceedingly numerous caries is rarely present". These deposits were also noted by Oxspring (1935) and Wilkinson (1953) who suggested that they were boney protruberances from the subchondral bone of the fibrocartilage surface.

Oxspring (1935) recorded his observations from post mortem examination of 128 feet from horses with navicular disease; "The pathological appearance of these structures varies enormously according to the progress of the disease". He suggested a progression of pathological changes, the first observable lesion being minute erosion of the fibrocartilage with concomitant rarefaction of the underlying compact bone. Often no tendon changes were present at this stage but in some cases there was a slight roughening of tendon fibres. These erosions, he maintained, developed into ulcers of the fibrocartilage, accompanied by further rarefaction of the subchondral bone, and tearing of the tendon fibres of the deep flexor tendon in an upward direction from a point opposite the distal border of the navicular bone. Oxspring (1935) also observed that the tendon lesions were located exactly opposite the fibrocartilage lesions and in no case had tendon lesions occurred without lesions being present on the fibrocartilage surface of the navicular bone. From these observations

he considered that the disease originated in the bone and that the tendon lesions were secondary, as a result of friction. The next stage in the development of the disease, was the collapse of areas of rarefied subchondral bone at the base of the fibrocartilage ulcers. This collapse resulted in the formation of cavities which, when formed, showed no tendency to increase in size, a point also noted by Hickman (1964).

Following cavity formation, in most cases adhesion of tendon fibres to the navicular bone occurred, thus restricting movement between the two surfaces (Oxspring, 1935). Wilkinson (1953) observed that adhesions between tendon and bone were always found when cavities were present in the navicular bone, although Smith (1886) and Oxspring (1935) found cavities in the navicular bone without tendon adhesions being present.

Brauell (1846), Smith (1886), Oxspring (1935) and Wilkinson (1953) all noted that these cavities occurred almost exclusively on or around the central ridge of the fibrocartilage surface of the navicular bone. It was suggested that this localisation of the cavities to the area around the central ridge was because of the vascular arrangement in the area combined with greater pressure from the deep flexor tendon on the sides of the ridge.

Hickman (1964) reported post mortem findings similar to those of Smith (1886) and Oxspring (1935). In addition he noted variations in the pathological changes in cases showing the typical clinical signs of navicular disease. For example, he found minute erosions of the fibrocartilage without rarefaction of the underlying compact bone in some cases, while in others he found rarefaction and cavitation of the underlying bone without any erosions of the fibrocartilage.

Both Oxspring (1935) and Hickman (1964) recorded osteophyte development on the medial and lateral extremities of the proximal border of the bone (spurs), in 10% of navicular disease cases.

Colles (1982) carried out post mortem examinations of the navicular bones of 100 normal control horses and 29 horses with navicular disease. The gross pathological findings which Colles (1982) noted as statistically significant in navicular disease were; brown discolouration and thinning of the fibrocartilage surface of the navicular bone; bone necrosis and tendon adhesions, and fractures of the navicular bone.

Reeks (1925), Oxspring (1935) and Wintzer (1964) reported that with increased pathological change the synovial fluid in the navicular bursa was reduced.

HISTOPATHOLOGY

Smith (1886) was the first to describe the "normal and pathological histology of the navicular bursa". The major changes he described in the navicular bone with navicular disease were in the blood vessels. The blood vessels increased in size and number in the subchondral bone of the fibrocartilage surface producing a rarefaction of the bone. He considered that when areas of the subchondral bone had been destroyed in this way, the fibrocartilage collapsed and was removed, thus forming the cavities described previously. Similar vascular changes were also reported by Wilkinson (1953) and Wintzer (1964). Wintzer (1964) maintained that the first changes occurred in the subchondral bone of the fibrocartilage and were followed by changes in the cartilage. Wilkinson (1953) placed more importance on the changes which occurred around the foramina

and vascular channels in the distal border of the navicular bone. He reported that these channels were filled with dilated sinusoids surrounded by granulation tissue, which extended further into the substance of the bone as the disease progressed. He also noted thrombi in some of the sinusoids, and thickening of the walls of the small arteries and blood vessels in the granulation tissue.

O'Brien et al. (1975) confirmed Wilkinson's (1953) findings, but considered the changes around the vascular channels to be secondary to chronic hyperaemia in the subchondral bone of the fibrocartilage surface.

Histological changes in the fibrocartilage of the navicular bone in navicular disease were reported by Smith (1886), Wilkinson (1953), Winzter (1964) and Colles (1982). O'Brien et al. (1975) examined the lesions on the fibrocartilage surface of the navicular bone using a correlative study with light microscopy and scanning electron microscopy.

The histological changes in the deep flexor tendon in navicular disease have been described by Smith (1886) and Wilkinson (1953). Smith (1886) considered that in navicular disease the tendon became fatty, and both he and Wilkinson (1953) noted the presence of "cartilage type cells" in the tendon. Smith (1886) believed these cells to be derived from the fibrocartilage of the navicular bone by friction, while Wilkinson (1953) maintained that they originated from the peritendonium of the bursal surface of the deep flexor tendon. Wilkinson (1953) reported that he could find no evidence of reparative processes in the affected deep flexor tendon.

PATHOGENESIS

It has been considered by many workers that mechanical/traumatic factors such as concussion, compression, and tension from ligaments were involved in the pathogenesis of navicular disease (Percivall, 1873; Smith, 1886; Fitzwygram, 1894; Oxspring, 1935; Wintzer, 1964; Adams, 1974 and others). Rooney (1974) stated that navicular disease was an arthrosis which developed on the fibrocartilage surface of the navicular bone and deep flexor tendon due to vibration and friction. The alterations in the fibrocartilage surface of the navicular bone reported by O'Brien et al. (1975) were, they considered, "attempts by the navicular bone to accomodate to mechanical stress".

Walley in 1885 suggested that navicular disease may be caused by a disturbance in the intra-osseous circulation of the navicular bone. Several workers since then have reported changes in the vasculature of the navicular bone in navicular disease (Smith, 1886; Wilkinson, 1953; Olsson, 1954; Hickman, 1964; Nemeth, 1972).

Post mortem angiography has been used to examine the arterial supply of the navicular bone in normal horses (Smith, 1886; Colles and Hickman, 1977) and in horses with navicular disease (Nemeth, 1972; Colles and Hickman, 1977). Colles (1982) found that in navicular disease cases there was an overall increase in the vascularisation of the navicular bone. There was dilation of sections of the distal arteries in the navicular bone and some of these arteries were completely or partially occluded. Histological examination of these occlusions showed thrombosis and arteriosclerosis of the vessels. Although Colles (1982) also found thrombosed arteries in the navicular bones of a small number of clinically normal horses, it was not a consistent finding, and he considered this to be a normal process in

some adult horses. In no case did he find macroscopic lesions in the navicular bone in the absence of thrombosis. He concluded that arterial thrombosis resulted in, or predisposed to, the pathological changes found in navicular disease. Histological examination of the navicular bones from the navicular disease cases showed evidence of bone necrosis, which he considered was due to localised ischaemia.

Nemeth (1972) also found thrombosed arteries in the navicular bones of navicular disease cases, which he related to filariasis.

Using barium sulphate angiography on dissected limbs from normal horses and navicular disease cases, Jones(1938) found thrombosis of sections of the medial digital arteries, but could find no connection between this lesion and navicular disease. Scott, Thrall and Sandler (1976) ligated the medial palmar and medial digital arteries in 7 shetland ponies and studied the effect using angiography over a period between 9 and 107 days. No lameness or changes in tissue viability resulted, and they concluded that both pre-existing and newly formed collateral vessels were sufficient to maintain viability of the limb.

Fricker, Reik and Hugelshofer (1982) examined angiographically the digital arteries of normal horses and horses with navicular disease. In all 10 horses with navicular disease examined, occlusions, partial or complete, were present in the digital arteries. They suggested that where occlusion extended to below the level of the artery connecting the medial and lateral digital arteries, on the posterior aspect of the fetlock, there would be a decreased blood flow through the foot. Colles (1982) demonstrated that in navicular disease cases there was a marked decrease in blood flow through the foot, which he considered could predispose to thrombosis.

Fricker et al. (1982) concluded that navicular disease was primarily a disease of the blood vessels and should be called "Endarteritis Obliterans equi".

Østblum, Lund and Melsen (1982) carried out histological examination of the navicular bones from 8 horses with navicular disease. Two of these 8 horses were double labelled with tetracycline as an intra-vital fluorescent bone marker (Frost, 1969). They could find no evidence of bone necrosis as reported by Colles (1982), and although arterial walls were often thickened, they found no occlusions or thrombosis. Histologically, they found a high rate of bone remodelling, and this was confirmed by fluorescent microscopy of sections from the bone labelled navicular bones, which showed a high level of tetracycline uptake. From this Østblum et al. (1982) concluded that ischaemic necrosis was not the primary cause of navicular disease, but suggested that increased pressure from the deep flexor tendon on the navicular bone as a result of conformational abnormalities of the limb, stimulated the increased bone remodelling.

INTRODUCTION

In this study clinical criteria have been used initially to diagnose navicular disease, followed by radiographic confirmation. It was considered important to examine the pathology in some of the cases, to assess whether the clinical signs were the result of the same or different pathological processes, and to compare the results of this examination with the results of a similar pathological examination of some of the control horses.

A correlative pathological study of the navicular bones and surrounding structures of 32 of the confirmed cases of navicular disease and 30 of the control cases was undertaken.

This study included, gross pathology, radiography post-mortem, angiography post-mortem, fluorescent microscopy following the administration of intra-vital fluorochrome bone labels, and light microscopy.

CHAPTER 4

PART I

GROSS PATHOLOGY

RADIOGRAPHY POST-MORTEM

ANGIOGRAPHY POST-MORTEM

GROSS PATHOLOGY

MATERIALS AND METHODS

Post-mortem examination was carried out on 32 horses with navicular disease, and 30 control horses, the navicular bones and adjacent portion of the deep flexor tendon were carefully dissected from both fore feet, and in some cases also from the hind feet. The gross pathological lesions present in the navicular bone and the adjacent portion of the deep flexor tendon were recorded, and the differences in incidence of the lesions between the groups were tested for significance by chi-squared analysis.

RESULTS

The results of gross pathological examination of the fore navicular bones and adjacent deep flexor tendon of 30 control horses, can be found in Appendix 4.1., and of the fore navicular bones and adjacent deep flexor tendons of 32 horses with navicular disease, can be found in Appendix 4.2. The results of examination of the hind navicular bones and adjacent deep flexor tendons of control horses, and horses with navicular disease of the hind feet, can be found in Appendices 4.3 and 4.4 respectively.

The gross pathological lesions found in the navicular bones have been classified into 8 categories (TABLE 4.1). All of these lesions, apart from fractures, are confined to the fibrocartilage surface of the navicular bone. The gross pathological lesions found in the portion of the deep flexor tendon adjacent to the navicular bone have been classified into 6 categories, most of which involve the surface of the deep flexor tendon which contacts the navicular bone (TABLE 4.2).

The gross pathological findings in the navicular bone and deep

flexor tendons were:

- (i) No visible lesion (N.V.L.) - when there was no evidence of any change in the fibrocartilage surface of the navicular bone, and/or on the adjacent surface of the deep flexor tendon, then N.V.L. was recorded (FIG 4.1). The N.V.L. category was recorded exclusively in the control group.
- (ii) Discolouration of the fibrocartilage surface of the navicular bone. This was a faint orange/brown discolouration of small areas of fibrocartilage, particularly in the region of the central ridge (FIG 4.2). The discolouration was often accompanied by a slight surface erosion of the fibrocartilage often only identified by the use of a hand lens, and by a similar discolouration in the corresponding area of the deep flexor tendon. This discolouration was rarely accompanied by erosion of the deep flexor tendon. A significantly higher incidence of discolouration of the fibrocartilage surface was found in the control group than in the navicular disease group. No significant difference was found in the incidence of discolouration of the deep flexor tendon between the 2 groups.
- (iii) Yellow discolouration of the fibrocartilage surface of the navicular bone (FIG 4.3): This lesion presented as a distinct yellow discolouration, distributed either in patches in the region of the central ridge, or diffusely over most of the fibrocartilage surface. The lesion was frequently accompanied by erosion of the fibrocartilage. In the control group, 50% of the navicular bones with yellow discolouration also had erosion of the fibrocartilage, and in the navicular disease group, all of navicular bones with yellow discolouration

also had erosion of the fibrocartilage.

A highly significant incidence of yellow discolouration of the fibrocartilage was found in the navicular disease group.

(iv) Yellow discolouration of the deep flexor tendon (FIG 4.3).

Many cases in which there was yellow discolouration of the fibrocartilage of the navicular bone also had similar lesions in corresponding areas on the surface of the deep flexor tendon. In some cases the yellow discolouration extended beyond the surface into the depth of the deep flexor tendon.

Ninety-six percent of the navicular disease cases with yellow discolouration also had erosion of the deep flexor tendon.

A highly significant incidence of yellow discolouration of the deep flexor tendon was found in the navicular disease group.

(v) Erosion of the fibrocartilage surface of the navicular bone (FIGS 4.3 and 4.4).

Erosion of the fibrocartilage was found usually in the region of the central ridge and often accompanied by yellow discolouration. Erosion of the fibrocartilage was recorded, without yellow discolouration being present, in navicular bones of both the control and navicular groups.

This lesion was recorded in 11(18.3%) of the navicular bones in the control group and in 57(95%) of the navicular bones in the navicular disease group. This was the most common lesion found in the navicular disease group and had a highly significant incidence.

(vi) Erosion of the deep flexor tendon. Erosions were found on the surface of the deep flexor tendon, usually in areas

corresponding to eroded areas of the fibrocartilage surface of the navicular bone (FIGS 4.3 and 4.4). This lesion had a highly significant incidence in the navicular disease group.

- (vii) Severe erosion of the fibrocartilage surface of the navicular bone (FIG 4.5).

Severe erosions were found in the region of, and involving, the central ridge of the fibrocartilage surface. As a result of severe erosion of the fibrocartilage, the underlying bone became visible as a red/brown discoloured area. This lesion was found only in the navicular disease group.

- (viii) Linear disruption of fibres of the deep flexor tendon. In this lesion the deep flexor tendon was eroded to the extent that small bundles of superficial tendon fibres were detached from the body of the tendon usually at the level of the distal border of the navicular bone and extending upwards (FIG 4.5). These lesions were always found a short distance medial or lateral to the central depression of the surface of the deep flexor tendon which accommodates the central ridge of the navicular bone. This lesion was found only in navicular disease cases.

- (ix) Small elevations on the fibrocartilage surface of the navicular bone (FIG 4.6).

These were small white raised areas about the size of a pinhead, found on the fibrocartilage surface of the navicular bone in areas where there was severe erosion of the fibrocartilage. They were only found in the navicular disease group, and in each case other pathological lesions were present.

- (x) Cavities in the fibrocartilage surface of the navicular bone

(FIG 4.7).

This lesion was found exclusively in the navicular bones of the navicular disease group. A single large cavity was present in 10 navicular bones, and in 4 navicular bones several small cavities were present. The cavities were located in, or in the region of, the central ridge of the fibrocartilage surface of the navicular bone.

In each instance in which cavities were found, these were in addition to other pathological lesions.

- (xi) Necrosis of the deep flexor tendon. Necrosis of the deep flexor tendon was considered to be present when there was severe erosion and disruption of a large number of tendon fibres. In 2 cases there was complete longitudinal separation of the deep flexor tendon (FIG 4.8).

Necrosis of the deep flexor tendon was found only in the navicular disease cases, and was always accompanied by other severe pathological changes.

- (xii) Adhesions between the deep flexor tendon and the navicular bone. The adhesions were in the form of a band, or bands of tendon fibres which were adherent to the navicular bone through cavities in the fibrocartilage surface (FIG 4.9). Adhesions between the deep flexor tendon and the navicular bone were found only in the navicular disease cases, and only in cases in which cavities were present in the fibrocartilage surface of the bone. In some instances cavities were found in the fibrocartilage surface of the navicular bone without adhesions.

- (xiii) Fracture of the navicular bone. This was found in 1 navicular bone in the navicular disease group. The fracture was a

complete vertical fracture extending from the proximal to distal borders, 1 cm lateral to the central ridge. No evidence of a bone callus was found and the 2 fragments were joined by a fibrous union.

(xiv) Depression in the fibrocartilage surface of the navicular bone.

A depression was found in the fibrocartilage surface of the navicular bones in 3 horses from the control group, 2 bilaterally and 1 unilaterally. A similar depression was found in 1 navicular bone in 1 horse from the navicular disease group.

In each case the depression was across the central ridge, midway between the proximal and distal border, and ranged from 5 mm to 15 mm in length, 2 to 5 mm in width, and in each case was approximately 1 mm in depth. No discolouration, erosion or thinning of the fibrocartilage was noted, on gross examination, in association with these depressions.

A similar distribution of lesions was found in the hind navicular bones and deep flexor tendons of the control horses as in the fore navicular bones and deep flexor tendons of the control horses.

The lesions recorded in the hind navicular bones and deep flexor tendons of the horses with hind limb navicular disease were similar to those found in fore limb navicular disease, except that adhesions between the deep flexor tendon and the navicular bone, and necrosis of the deep flexor tendon, were not found (FIG 4.10).

DISCUSSION

Hickman (1964) considered that navicular disease was not a specific entity, but that the clinical signs were the result of different pathological processes in the navicular bone, and Colles (1982) defined navicular disease pathologically as any condition affecting the navicular bone and surrounding structures.

In 1892, Bryden maintained that, "to regard degeneration of the navicular bone, as met with in true navicular disease, as the same disease of the same bone, the result of inflammation of a nail having accidentally or otherwise penetrated it", was fundamentally wrong.

The differences in opinion which exist on the pathology and pathogenesis of navicular disease may be due partly to a lack of clear disease definition, and partly to difficulties in assessing the temporal relationship of the various pathological changes found in the navicular bone and surrounding structures in navicular disease cases.

The diagnosis of navicular disease is based on finding certain clinical signs in the horse, and then confirmed by radiography of the navicular bone. The pathological processes, must therefore, have developed to a stage where clinical and radiographic changes were manifest, before the disease could be diagnosed. This combined with the economics of the horse industry, which dictate that a horse with navicular disease is not usually destroyed until the disease has reached an advanced stage, means that the opportunities to examine the course of the disease, and to assess which are the primary pathological lesions and which are secondary, are limited.

Colles (1982) pointed out that very few of the pathological studies of navicular disease also include material from control horses.

It is important to establish, particularly in joints which are the subject of constant pressures, the degree of pathological change which is associated with 'normal' wear and tear.

The gross pathological examination in the present study showed that in all the cases of navicular disease macroscopic changes were evident in the navicular bones and contiguous deep flexor tendon, and in 43% of the control cases, macroscopic changes were evident in the navicular bone and/or deep flexor tendon.

The gross pathological lesions found were:

Discolouration of the fibrocartilage surface of the navicular bone and the deep flexor tendon

The majority of the pathological lesions which were found in the control cases were discolouration of the fibrocartilage surface, which in most cases was accompanied by a similar discolouration on the surface of the deep flexor tendon. Forty-two percent of the control cases with discolouration of the fibrocartilage also had erosions of the fibrocartilage, but erosions accompanying discolouration of the deep flexor tendon were not found to the same extent.

Colles (1982) considered yellow discolouration and erosion of the fibrocartilage surface of the navicular bone, to be a normal wear lesion, since he had found no significant difference in the incidence of this lesion between the control and navicular disease cases.

In this present study a distinction has been drawn between

faint orange/brown patches of discolouration which had a significant incidence in the control group, and distinct yellow discolouration of the fibrocartilage surface of the navicular bone which had a highly significant incidence in the navicular disease cases.

From these findings it is suggested that the orange/brown discolouration and erosion of the fibrocartilage of the navicular bone may correspond to the normal wear lesions described by Colles (1982). The apparent significant incidence of this lesion in the control cases may not be correct, because the low incidence of this slight discolouration in navicular disease cases is possibly due to the other pathological lesions present masking any slight change.

The macroscopic appearance of the patches of orange/brown discolouration and erosion of the fibrocartilage surface of the navicular bone resembles closely, lesions of the articular cartilage of other joints in the horse, which were not necessarily of clinical significance (Fraser, 1983).

The areas of orange/brown discolouration on the surface of the deep flexor tendon correspond to those on the fibrocartilage surface of the navicular bone. In no case was discolouration of the deep flexor tendon found without discolouration of the fibrocartilage surface. Erosion of the deep flexor tendon accompanying the discolouration was found in fewer of the control horses than was erosion of the fibrocartilage surface. It is likely therefore that the slight discolouration and erosion of the deep flexor tendon is secondary to changes in the fibrocartilage surface of the navicular bone.

Yellow discolouration of the fibrocartilage surface of the navicular bone and the adjacent deep flexor tendon

This discolouration was quite different to that described before. These lesions are similar to those described by Oxspring (1935), Wilkinson (1953), and Olsson (1954). Wilkinson (1953) maintained that yellow discolouration was of no significance in navicular disease and was found mainly in older horses. In this present study yellow discolouration on the fibrocartilage surface of the navicular bone, and on the adjacent deep flexor tendon, was found in horses from 5 to 16 years old, and therefore was not predominantly a feature of older horses.

In all of the horses used in this study, other joints including the pedal, fetlock and carpal joints were also examined. Yellow discolouration similar to that in the navicular bone and deep flexor tendon was not present. Webbon (1977) described yellow discolouration and yellow pigment spots in the superficial flexor tendon at the level of the proximal sesamoid bones, and in the deep flexor tendon, proximal to the proximal sesamoid bones and at the level of the navicular bone.

The extent of the yellow discolouration of the fibrocartilage and deep flexor tendon did not appear to be related to the severity of pathological change.

Yellow discolouration of the deep flexor tendon was not found in the absence of yellow discolouration or severe erosion of the fibrocartilage of the navicular bone.

Erosion of the fibrocartilage surface of the navicular bone and of the deep flexor tendon

These lesions had a significantly high incidence in the navicular disease cases, although they were present in some of the control horses.

The erosions in most cases accompanied yellow discolouration, and were found in the region of the central ridge and corresponding area on the deep flexor tendon.

Severe erosion of the fibrocartilage surface of the navicular bone

This lesion was easily detected on the fibrocartilage surface of the navicular bone, since it was visible as a well demarcated area of red/brown discolouration. The fibrocartilage in these cases was eroded to the extent that the underlying subchondral bone was visible, and the discolouration was considered to be due to congestion of this area of bone. The lesion was not present in any of the control cases, and when present in the navicular disease cases, this was in addition to other pathological changes. This severe erosion of the fibrocartilage surface of the navicular bone is similar to that described by O'Brien et al. (1975) in navicular disease cases.

Linear disruption of the deep flexor tendon

Linear disruption of the deep flexor tendon was well described by Wilkinson (1953). He suggested that small bundles of tendon fibres may have been adherent to the fibrocartilage surface of the navicular bone, and with movement, have been torn away and "curled upwards due to their own elasticity".

Linear disruption of the deep flexor tendon in this present

study was in addition to and directly opposite severe erosions of the fibrocartilage surface of the navicular bone, in all but one case. However, severe erosion of the fibrocartilage surface was present in some navicular disease cases without linear disruption of the deep flexor tendon.

Colles (1982) showed that during a normal stride the navicular bone moved considerably, thus allowing it to absorb concussion. It therefore seems likely that the linear disruption of the deep flexor tendon is secondary to severe erosion of the fibrocartilage surface of the navicular bone, and is brought about by the movement of the navicular bone and deep flexor tendon producing upward stripping of tendon fibres.

Small elevations on the fibrocartilage surface of the navicular bone

These small, raised, white areas receive little mention in the literature, but have been described as calcareous deposits by both Smith (1886) and Oxspring (1935), and as boney protuberances by Wilkinson (1953).

In this present pathological study, these were noted only in navicular disease cases and only in cases in which there was also severe erosion of the fibrocartilage surface of the navicular bone. This may be because these small elevations only develop at the same time as severe erosion of the fibrocartilage, or they develop before this stage and only become evident on gross examination with thinning of the surrounding fibrocartilage.

Smith (1886) considered that these calcareous deposits were rarely found when cavities were present in the fibrocartilage surface of the navicular bone. This was not the finding in this

present study, and 13 out of the 14 navicular bones with cavities in the fibrocartilage surface also had small elevations present.

Cavities in the fibrocartilage surface of the navicular bone, and adhesions of the deep flexor tendon to the navicular bone

Cavities were always located in the region of the central ridge of the navicular bone. Adhesions of the deep flexor tendon to the fibrocartilage surface of the navicular bone were found in over half the cases in which cavities were present. However, tendon adhesions were not found in the absence of cavities.

The mechanisms whereby adhesions are formed between the deep flexor tendon and the navicular bone are poorly understood. Wilkinson (1953) considered that adhesions developed as a result of granulation tissue within the navicular bone proliferating through the cavity in the fibrocartilage surface, and coming in contact with the damaged deep flexor tendon. Webbon (1977) noted that in the digital sheath, where the fibres of the deep flexor tendon are not separated by paratenon from the surrounding synovial fluid, healing of damaged tendon fibres is prevented by a rapid growth of synovial cells over the damaged area. This situation also applies to the deep flexor tendon at the level of the navicular bone, and it is possible that adhesions develop due to a combination of proliferating vascular granulation tissue and a reduction of healing capacity on the part of the damaged deep flexor tendon.

Necrosis of the deep flexor tendon

Necrosis of the deep flexor tendon was also found in conjunction with cavities in the fibrocartilage surface of the navicular bone.

It is suggested that necrosis of the tendon occurs as a result of constant trauma from the damaged fibrocartilage surface combined with a reduction in healing capacity on the part of the tendon. It is also possible that necrosis of the deep flexor tendon may occur in some cases following rupture of adhesions between the tendon and the navicular bone.

Fracture of the navicular bone

In this pathological study only one fractured navicular bone was found. The fracture was typical of a non-union fracture. A non-union fracture is defined by Hickman (1964) as one in which healing process ceases, and the gap between the fracture fragments is filled with fibrous tissue.

The factors which can lead to a non-union fracture are, inadequate immobilisation, hyperaemia or inadequate blood supply, or concurrent pathological processes.

Post-mortem examination of a number of fractured navicular bones of horses in which there had been no evidence of concurrent navicular disease clinically or radiographically, showed fractures of a similar non-union type as in the navicular disease case. It is therefore considered that the concurrent pathological processes of navicular disease do not necessarily cause non-union of fractures.

Depression in the fibrocartilage surface of the navicular bone

The navicular bones in which depressions in the fibrocartilage surface had been found showed no evidence of other gross pathological changes, either in the fore or hind feet.

The depressions are similar to those described by Colles (1982), which he considered to be congenital and unrelated to navicular

disease.

In all the cases of navicular disease examined, gross pathological changes were present in the navicular bones and adjacent portion of the deep flexor tendon. The degree of pathological change present varied between cases, and was related to the progression of the disease and the stage at which the horse was destroyed.

It was noted that in cases of bilateral navicular disease the degree of pathological change was not the same in each fore foot. This finding concurs with that of Oxspring (1935) who concluded that this disparity in pathological change in cases of bilateral navicular disease was because the changes in one fore foot were secondary.

It is possible that the disease process does not start at the same time in both fore feet, or the disease process may be initiated in both fore feet at the same time but, because of other factors, it progresses faster in one fore foot than in the other.

In all of the cases in which there was hind limb navicular disease the gross pathological changes in these hind navicular bones and adjacent deep flexor tendons were always less severe than the changes present in the fore feet. This also suggests that either the disease process in the hind feet starts later than in the front feet, or starts at the same time and progresses at a slower rate.

The gross pathological change on the fibrocartilage surface of the navicular bone in this study was almost always located in the region of the central ridge. This observation was noted by a number of workers (Smith, 1886; Oxspring, 1935; Wilkinson, 1953; O'Brien et al., 1975). It was suggested by Oxspring (1935) that the localisation of the pathological lesions to the area around the central ridge was associated with the normal vascular arrangement

in the area combined with tendon pressure on either side of the ridge.

The pathological changes in the deep flexor tendon were almost always opposite, and mirrored in many cases, the changes on the fibrocartilage surface of the navicular bone. Gross pathological changes were not found in the deep flexor tendon without changes being present on the fibrocartilage surface of the navicular bone, although the reverse situation did occur. These findings suggest that the pathological changes in the deep flexor tendon are secondary to the changes in the fibrocartilage surface of the navicular bone.

RADIOGRAPHY POST-MORTEM

MATERIALS AND METHODS

Following gross pathological examination, the isolated navicular bones from the control and navicular disease groups were then examined radiographically.

The navicular bone was placed, articular surface uppermost, on the x-ray cassette, and angled so that the anterior and posterior edges of the distal border of the navicular bone were parallel. The navicular bone was positioned in this manner so that the extent and shape of the distal nutrient foramina could be appreciated, and in order to replicate as far as possible the position of the navicular bone in the foot during routine radiography, using the upright pedal route with the hoof wall at 80° to the horizontal.

The radiographs were taken using high definition screens*, fast film†, a 90 cm focus-film distance and the exposure factors ranged from 56-66 kV and 25-40 mAS.

Several antero-posterior views were taken of each navicular bone and the results recorded.

The findings recorded from the antero-posterior radiographs of the navicular bones were:

- (i) The number of nutrient foramina visible in the distal border of the bone.
- (ii) The shapes and position of these distal nutrient foramina.
- (iii) The navicular score for each bone.
- (iv) Increased radiodensity in the distal border and around the

* Kodak Ltd., London, England.

+ X-Omat S, Kodak Ltd, Manchester, England.

· nutrient foramina in the distal border.

- (v) The presence of nutrient foramina in the proximal border of the bone.
- (vi) Spur formation.
- (vii) Bone growth on the proximal border of the navicular bone.
- (viii) Areas of radiolucency in the body of the navicular bone.
- (xi) Change in trabecular pattern. Change in the pattern of trabeculae in the medulla of the navicular bone in cases of navicular disease was suggested as significant by Van der Watering (cited by Kealy, 1972), while O'Brien et al. (1975) described a loss of trabecular pattern in navicular disease cases. In this present study, a distinct, uniform trabecular pattern in a radiograph of a navicular bone, has been recorded as a negative, and a disorganised trabecular pattern as a positive.

The radiographic findings of the antero-posterior view of the navicular bones post-mortem were compared to those obtained from the antero-posterior views of the bones in situ.

Both fore navicular bones of 10 of the control cases, and the affected fore navicular bones of 10 of the navicular disease cases, were then cut into 4 sections, 1-4, as shown in FIG 4.12.

Radiographs were taken of each section, using a lateral view. The same high definition screens, film, focus-film distance and exposure factors were used, as in the antero-posterior radiographs of the navicular bones.

The results were analysed statistically for significance using chi-squared distribution.

RESULTS

The findings from radiographic examination of the antero-posterior views of the navicular bones from the control cases are contained in APPENDIX 4.5, and from the navicular disease cases in APPENDIX 4.6.

The following additional radiographic changes were noted in the radiographs of the isolated navicular bones when compared to the radiographs taken as part of the clinical diagnostic procedure.

(i) Number and position of distal nutrient foramina

An additional type A foramen was present in 10 out of the 60 isolated navicular bones from the control group, and an additional type A or B foramen was found in 10 navicular bones from the navicular disease group.

(ii) Increased radiodensity

Increased radiodensity of the distal border of the navicular bone was recorded in 8 navicular bones in the control group, and 1 navicular bone in the navicular disease group.

(iii) Nutrient foramina in the proximal border

Nutrient foramina were visible in the proximal border of 5 navicular bones in the control group, and 14 navicular bones in the navicular disease group.

(iv) Spurs

One navicular bone in the control group and 1 navicular bone in the navicular disease group had small spurs, which had not been identified in the routine radiographs.

(v) Bone growth on the proximal border

This feature was found in 5 of the navicular bones in the control group and 1 navicular bone in the navicular disease group.

There was no difference between the radiographs of isolated navicular bones, and the routine radiographs of the navicular bone, in the following features:

- (i) The shape of the distal nutrient foramina in both the control and navicular disease group.
- (ii) Radiolucent areas in the medulla. This feature was exclusive to the navicular bones of the navicular disease group.

A radiographic feature recorded from radiographs of the isolated navicular bone, but not from routine radiographs of the navicular bone, was a change in trabecular pattern.

Change in trabecular pattern

Forty-eight (80%) of the navicular bones from the control group had a uniform trabecular pattern, the trabeculae arranged in rows parallel to the distal border of the bone (FIG 4.13). This uniform trabecular pattern was also found in 15 (25%) of the navicular bones from the navicular disease group, but in the remaining navicular bones of this group the trabeculae were arranged in a more irregular manner (FIG 4.14).

Loss of the uniform trabecular pattern was found in a highly significant number of navicular bones in the navicular disease group. ($\chi^2 = 34.2$; d.f. = 1; $P < 0.001$)

In a number of the navicular bones, in which there was a loss of uniform trabecular pattern, increased density and width of some

of these trabeculae, particularly around areas of radiolucency, was noted.

The results of radiographic examination of the sections of navicular bones, from the control group and navicular disease group, are contained in APPENDICES 4.7 and 4.8 respectively, and the results of statistical analysis in TABLE 4.3 . Most of the radiographic features were recorded from sections 2, and 3 (FIG 4.12).

The radiographic features were:

- (i) Change in the navicular bone outline. Morgan.(1972) and Klessinger (1973) considered that, on a lateral view, the outline of the normal navicular bone was square or slightly trapezoid, and that, in navicular disease, remodelling took place, so that the outline became markedly trapezoid or triangular.

In this study 18(90%) of the navicular bones in the control group had a square outline, and 9(50%) of the navicular bones in the navicular disease group had a square outline (FIG 4.15). The remaining 2(10%) navicular bones in the control group were trapezoid in outline, and the 9(50%) in the navicular disease group, a significant number, were markedly trapezoid or triangular in outline (FIG 4.16).

- (ii) Loss of subchondral bone of the fibrocartilage surface of the navicular bone. In this study this feature was found only in the navicular bones from the navicular disease group and only in sections 2 and 3. In most cases, the loss of subchondral bone was localised to the central part of the fibrocartilage surface, and varied in degree from a slight thinning at the cartilage surface to complete loss of a section of bone plate

and underlying trabeculae (FIGS 4.17 - 4.20).

- (iii) Areas of radiolucency in the subchondral bone of the fibrocartilage surface. This lesion was in the form of small radiolucent streaks or patches in the subchondral bone plate. It was found in 4(20%) of the navicular bones in the control group, and 14(77.7%), a significantly higher number in the navicular disease group (FIGS 4.18 and 4.19).
- (iv) Change in trabecular pattern. A pattern, in which trabeculae ran in lines from the articular to fibrocartilage surface of the navicular bone, parallel to the distal border of the navicular bone, was noted in all of the navicular bone sections from the control group, but only in 4(22%) of the navicular disease group. The other 14 navicular bones in the navicular disease group showed a loss of uniform trabecular pattern, the trabeculae being arranged in a more disorganised manner.
- (v) Increased density and width of trabeculae. This was a radiographic feature found only in the navicular bones of the navicular disease group. These trabeculae were found mainly bordering areas of radiolucency (FIG 4.20).
- (vi) Bone growth on the proximal border. Bone growth was noted on the proximal border of 4 navicular bones in the control group, and 8 navicular bones in the navicular disease group. No significant difference was found between the 2 groups in the incidence of this lesion.
- (vii) Notch in the distal border. This was noted in 12 bones in control group and 11 bones in the navicular disease group. There was no significant difference in the incidence of this lesion, between the groups.

- (viii) Increased radiodensity in the distal border of the navicular bone. Increased radiodensity in the distal border of the navicular bone was present in 8(40%) of the navicular bones from the control group, and 14(78%) from the navicular disease group. A significant incidence of increased radiodensity in the distal border was present in the navicular disease group.
- (ix) Radiolucent areas in the navicular bone. This radiographic lesion was found exclusively in the navicular disease group, and was present in 7 out of the 18 navicular bones examined.

DISCUSSION

Most of the radiographic features found in the radiographs of the isolated navicular bones had previously been identified on routine radiographs of the navicular bone.

In some cases a type A or B foramen was found in the distal border of the isolated navicular bone, which had not been visible on the radiographs of the bone in situ.

Additional changes, which were found on the radiographs of a small number of isolated navicular bones, and which had not been visible on the routine radiographs were, increased radiodensity, nutrient foramina in their proximal border, spurs, and bone growth on the proximal border.

The finer details of the navicular bone ultrastructure, such as the pattern, density and width of trabeculae were not recorded in the routine radiographs. It was not possible to distinguish the trabecular pattern clearly in the routine radiographs, since the finer details of the trabeculae were lost because the shadow of the

navicular bone overlay that of the second phalanx. In spite of this, trabecular disruption was noted in some cases, and was always in the region of the central ridge.

In both the antero-posterior and lateral radiographs of the isolated navicular bones, the trabecular pattern was easily identified.

It is generally accepted that the trabeculae of cancellous bone are aligned along the principal stress direction, to provide the maximum structural support with the minimum of osseous tissue (trajectorial theory), although the mechanisms by which this is achieved are not fully understood.

The application of a force or load to a bone will cause the bone to deform. The deformation imposed on a bone during normal activity is termed "customary intermittent deformation" (Lanyon, 1982).

The orientation of trabeculae can be altered in response to changes in deformation. It is considered that intermittent deformation, as in normal activity, rather than static strain, is a stimulus to bone remodelling (Hert, Liskova and Landa, 1971). Lanyon (1982) suggested that customary intermittent deformation may be responsible for determining the arrangement and maintenance of bony structures. If the bone structure is not properly maintained, as in intercurrent disease, the normal intermittent deformation may then result in damage to bone structures.

The change in trabecular pattern recorded in the navicular disease cases could therefore be related to:

- (i) Changes in the normal stress on the navicular bone associated with altered weight bearing.

or to

- (ii) The pathological processes of navicular disease affecting

trabecular structure.

or to a combination of these.

The results of this present study indicate that a change in trabecular pattern, on routine radiographs of the navicular bone, is an important radiographic finding in navicular disease cases, although change in trabecular pattern is not always easy to detect on an antero-posterior view.

The lateral views of the navicular bone sections showed that a significantly higher number of the navicular disease cases had a markedly trapezoid or triangular outline. The change, from the normal square outline of the navicular bone to a triangular outline, may also be the result of remodelling processes as part of the adaptive response.

Most of the navicular bone sections, in which there was a change in bone outline, also had bone growth on the proximal border, and loss of subchondral bone on the fibrocartilage surface, both of these accentuating the triangular outline.

Loss of subchondral bone of the fibrocartilage surface was found only in the navicular bones from the navicular disease group. Different degrees of subchondral bone loss were recorded. In some cases there was complete loss of areas of the subchondral bone plate and loss of trabecular bone beneath it, (FIG 4.20), while in others there was only thinning of the subchondral bone plate, (FIG 4.18).

In most cases in which there was thinning of the subchondral bone plate of the fibrocartilage surface, there were also areas of radiolucency in this plate, which varied in extent. The amount of radiolucency increased as the degree of subchondral bone thinning increased, and in no navicular disease case, were these areas of

radiolucency found in the absence of subchondral bone thinning.

The results of this study indicate that, areas of radiolucency in, and thinning of the subchondral bone plate of the fibrocartilage surface of the navicular bone on lateral radiographs, are important findings in navicular disease cases. However, these findings together with change in trabecular pattern, are not always easy to detect on routine radiographs.

The navicular score for each of these sectioned navicular bones was then examined in relation to the degree of radiographic change in their subchondral bone plate (FIGS 4.17 - 4.20). It was found, from the small number of navicular bones examined, that the navicular score increased as the degree of radiographic change in the subchondral bone plate appeared to increase.

COMPARISON OF GROSS PATHOLOGY AND RADIOGRAPHIC FINDINGS POST-MORTEM

INTRODUCTION

The gross pathological changes in the navicular bone, found earlier in this study (APPENDICES 4.1 and 4.2) were mostly situated in the cartilage of the fibrocartilage surface, while radiographic lesions described in the previous section involved the subchondral bone plate of the fibrocartilage surface. When these radiographic changes in the subchondral bone plate were compared to the gross pathological lesions on the fibrocartilage surface of these bones, it was found that as the degree of gross pathology in the cartilage increased, so the degree of radiographic change appeared to increase.

In the previous section, a correlation was found between the degree of radiographic change in the subchondral bone plate of the fibrocartilage surface of a navicular bone, and its navicular score. Following these findings, it was decided to examine the relationship between the gross pathological changes on the fibrocartilage surface of each navicular bone, and its navicular score.

MATERIALS AND METHODS

The gross pathological changes on the fibrocartilage surface of the navicular bone and adjacent deep flexor tendon recorded earlier, (APPENDICES 4.1 and 4.2) were classified into 7 grades according to the degree of change present.

These grades were:

1. No visible lesion on the fibrocartilage surface of the navicular bone or on the deep flexor tendon (FIG 4.1).
2. Small areas of orange brown discolouration, on the fibro-

cartilage of the navicular bone, which may also be present on the deep flexor tendon. This lesion is often accompanied by slight erosion of the fibrocartilage (FIG 4.2).

3. Distinct yellow discolouration of the fibrocartilage surface of the navicular bone and deep flexor tendon, accompanied by erosion of the fibrocartilage and slight roughening of fibres on the surface of the deep flexor tendon (FIG 4.3).
4. Distinct yellow discolouration of the fibrocartilage surface of the navicular bone and deep flexor tendon, with marked erosion and some thinning of the fibrocartilage, and marked erosion of the deep flexor tendon (FIG 4.4).
5. Discolouration, marked erosion and thinning of the fibrocartilage, to the extent that the underlying bone becomes visible and in some cases, small bony elevations are evident. There is marked linear erosion of the deep flexor tendon, with tearing and upward curling of superficial fibres. (FIG 4.5).
6. Discolouration, severe erosion of the fibrocartilage exposing the underlying bone, in which small cavities are evident, accompanied by severe linear disruption of fibres of the deep flexor tendon (FIG 4.11).
7. Discolouration, severe erosion of the fibrocartilage exposing the underlying bone, in which a large cavity or cavities are present, with adhesions of the deep flexor tendon to the navicular bone, or without adhesions but necrosis of the deep flexor tendon (FIGS 4.8 and 4.9).

The fore navicular bones and deep flexor tendons of the 30 control horses, and the 32 horses with navicular disease, were then classified into one of the 7 grades, according to the degree of

change present.

The navicular score for each of these navicular bones was calculated from the antero-posterior radiographs of the navicular bones in situ.

The results of gross pathological examination were plotted against the navicular score for each bone in the control group (FIG 4.21), and in the navicular disease group (FIG 4.22), and the correlation coefficients calculated.

RESULTS

A significant positive correlation was found between the gross pathological changes and the navicular score of the navicular bones from the control group ($r = +0.299$; d.f. 58; $P < 0.05$), and a highly significant positive correlation was found between the gross pathological changes and the navicular score of the navicular bones from the navicular disease group ($r = +0.843$; d.f. 58; $P < 0.01$).

DISCUSSION

Several workers have related the gross pathological findings to the radiographic findings in navicular disease cases, (Oxspring, 1935; Olsson, 1954; O'Brien et al., 1975; Colles, 1982).

The 7 grades of gross pathological change affecting the fibrocartilage surface of the navicular bone and adjacent deep flexor tendon, are similar to the 4 groups of changes described by Olsson (1954). He examined the radiographic changes in macerated navicular bones in relation to their gross pathology, and found a definite correlation between them.

O'Brien et al. (1975) found a positive correlation between the radiographic lesions, using a dorso-palmar view of the navicular bone,

and the gross pathological and histological changes.

On all of the navicular bones from the navicular disease cases examined in this study, gross pathological lesions were found in the cartilage of the fibrocartilage surface of the navicular bone.

It has been suggested by numerous workers (Oxspring, 1935; Wintzer, 1964; O'Brien et al., 1975 and others) that the primary pathological changes occurred in the subchondral bone of the fibrocartilage surface. These changes presented histologically as thinning and osteoporosis of the subchondral bone plate.

In this present study radiographic changes, in the form of thinning and areas of radiolucency in the subchondral bone plate of fibrocartilage surface, were found in some of the navicular bones in the navicular disease group. In these navicular bones the navicular score increased as the degree of radiographic change in the subchondral bone plate increased.

When the navicular score was compared with the gross pathological findings in the navicular bones of the navicular disease group, a positive correlation was found. The navicular score was calculated from the antero-posterior views of the navicular bone in situ rather than from the isolated navicular bone, since very little difference in navicular score had been found between the two.

It is therefore considered from these findings that the navicular scoring system is a useful indicator of the possible degree of pathological change present in the navicular bone and deep flexor tendon in navicular disease cases. However, it is stressed that there is an overlap in the navicular scores of different grades of pathological change and therefore the navicular scoring system should not be used as certain evidence of the degree of pathological change

present.

A positive correlation was also found between the navicular scores and the degree of gross pathological changes present in the navicular bones of the control group.

ANGIOGRAPHY POST-MORTEM

MATERIALS AND METHODS

Angiography post-mortem was carried out in the fore and hind limbs of 4 horses from the control group, A9, A10, A44 and A71.

The same process was also carried out in the fore limbs of the 4 horses from the navicular disease group, B7, B12, B13, and B14.

The limbs were removed immediately after death at the level of the fetlock joint. The medial and lateral digital arteries were cannulated and flushed manually with approximately 2-3 litres of heparin saline, (10,000 i.u. of heparin* per 1 litre of physiological saline), or until such time as the heparin saline returning in the digital vein was free of blood. Following this, 20-30 mls of a suspension of 75% barium sulphate⁺ and, 25% methylene blue[#], was injected manually (Colles, 1982). Barium sulphate was used in the injection because it was radio opaque and could be visualised by radiography, and the methylene blue was used to enable the blood vessels to be identified, following Spalteholtz technique (Culling, 1963; Kelly, 1968).

The cannulas were removed, the digital arteries and veins occluded using artery forceps, and radiography of the navicular bones in situ was carried out using the routine method.

The navicular bones were then carefully dissected from the foot, the gross pathological changes recorded, and an antero-posterior radiograph of the bone taken using the method described in the previous section.

* Boots Company Limited, Nottingham, England.

+ Micropaque 1 gm/ml Nicholas Ltd, England.

May and Baker Ltd, Dagenham, England.

Following radiography, the navicular bone was placed in 10% buffered formal saline solution for 7-10 days for complete fixation. The bone was then transferred, intact, into Gooding Stewart's* solution for decalcification. The decalcifying solution was changed at regular intervals until decalcification was complete. After decalcification, the navicular bone was dehydrated through alcohols, cleared in Xylene, and then placed in Spalteholz solution. Spalteholz solution is composed of equal parts of benzyl benzoate⁺ and Oil of Wintergreen[#] (Culling, 1963). Once cleared, the navicular bone was examined under a dissection microscope. The navicular bone was then cut into 5 mm sections in the sagittal plane, or parallel to the distal border, and again examined under a dissection microscope. Radiography of these sections was carried out as before, using high definition screens, fast film, and exposure factors of 36 kV and 150 mAS.

RESULTS

Very little information was obtained from the radiographs of the undecalcified navicular bone, because the boney structures obscured the finer details of the vasculature. Blood vessels could be identified entering the navicular bone along the distal and proximal borders, but could only be followed for a short distance into the navicular bone.

* 10% formalin in 10% formic acid

+ May and Baker Ltd., Dagenham, England.

Hopkins and Williams, Chadwell Heath, Essex, England.

Following decalcification and clearing, 2 sources of blood supply were identified in the fore and hind navicular bones of the control horses using both the dissection microscope and radiography (FIGS 4.23 and 4.25). These sources of supply were in the proximal and distal borders, and extensive anastomoses were present between them. Numerous blood vessels radiated from these main sources to the subchondral bone of both the articular and fibrocartilage surfaces of the navicular bone. Blood vessel number and size was similar in the subchondral bone of both the articular and fibrocartilage surfaces. In the hind limb navicular bones the distribution of blood vessels was similar, but the vessels appeared to be more numerous, and of smaller diameter, than in the navicular bones of the fore limbs.

In all of the navicular bones examined in the navicular disease group there was a marked increase in the number of, and anastomoses between, the arteries within the navicular bone. Many of these vessels were dilated both in the medulla and in the subchondral bone plates of the navicular bone. This increased vascularisation was particularly evident in the distal border and in the subchondral bone of the fibrocartilage surface. (FIGS 4.24 and 4.26). A similar increase in vascularisation was not noted in subchondral bone of the articular surface.

Many of these blood vessels in the navicular disease cases were found to be convoluted.

DISCUSSION

The first report of microangiography of the navicular bone was by Smith in 1886. He examined the arrangement of the blood vessels

in the navicular bone, using Prussian blue dye, and concluded that the normal navicular bone was highly vascular, and in navicular disease there was an increase in size and number of blood vessels, particularly in the subchondral bone of the fibrocartilage surface.

Microangiography has been used by numerous workers since then in the investigation of navicular disease (Vaughan, 1961; Wintzer, 1964; Colles and Hickman, 1977; Colles, 1982; Svalastoga, 1983).

The methods used in this study, to inject and examine the navicular bones, are similar to those described by Colles and Hickman (1977) and Colles (1982). The navicular bones, having been injected with a suspension of contrast medium and dye, were dissected from the foot and examined by radiography. Little information on the vascular pattern within the navicular bone was obtained from these radiographs. This is similar to the findings of Wintzer (1964) and Svalastoga (1983), who concluded that the bone must first be decalcified before the detailed vascular pattern can be traced.

The results of examination of the vascular arrangement, by dissection microscope and by radiography, of the control navicular bones, confirm the findings of Colles and Hickman (1977), Colles (1982) and Svalastoga (1983).

Microangiographic examination of hind navicular bones was only carried out in the control horses. The results of these examinations showed that, although the distribution of blood vessels in the hind navicular bones was similar to that in the fore navicular bones, the blood vessels appeared to be present in greater numbers in the hind navicular bones than in the fore navicular bones. In general, these vessels in the hind navicular bones appear to be smaller in diameter than in the fore navicular bones.

The greater number of blood vessels found in the hind navicular bones in this work is in accord with the work of James et al. (1980), who found that there was a greater average number of arteries entering the distal border of the hind navicular bones than in the fore navicular bones.

All of the navicular bones in the navicular disease cases examined showed an increase in size, number, and anastomoses of the blood vessels. In the navicular bones of the control horses no difference in the distribution and extent of vascularisation was found between the subchondral bone of the fibrocartilage and articular surfaces. However, in the navicular bones of the navicular disease cases, there was a marked increase in the vascularisation of the subchondral bone of the fibrocartilage surface, which was not evident in the subchondral bone of the articular surface. The vascularisation was increased as a result of an increase in both the size and number of blood vessels present. These findings are in accord with the work of Smith (1886) and Svalastoga (1983).

Colles and Hickman (1977) and Colles (1982) reported, from microangiographic studies of the navicular bone, the presence of vascular occlusion of arteries in the distal part of the bone in navicular disease cases. This present study failed to confirm these findings, since no evidence of vascular occlusion was found in any of the navicular bones examined.

The results of the present microangiographic study are similar to those of Harrison, Schajowicz and Trueta (1953), who found an increase in the number and size of the blood vessels in osteoarthritic femoral heads in man. Although it had been suggested by a number of workers that ischaemia was involved in the development

of osteoarthritis (Phemister, 1940; Pridie, 1952; and others), Harrison et al. (1953) maintained that the increased vascularisation, found in their angiographic studies, was not a reaction to an earlier ischaemic episode.

The gross pathological findings in these navicular bones was compared with the microangiographic findings.

In all of the navicular bones in the navicular disease cases there was discolouration, and severe erosion of the fibrocartilage, exposing the underlying bone. The red/brown discolouration, found when the fibrocartilage was severely eroded, was thought to be due to congestion of the underlying subchondral bone. The results of the microangiographic study confirm this finding, since the area in which there was severe cartilage erosion corresponded to the area of the subchondral bone in which there was greatest increase in vascularisation.

Of the navicular bones examined from the control group, 2 had no visible lesions on the fibrocartilage surface, and the remaining 6 had slight orange/brown discolouration of the fibrocartilage surface. Although only a small number was examined, no difference was noted in the vascularisation of the subchondral bone of the fibrocartilage surface between the control navicular bones with no visible lesion, and the control navicular bones with orange/brown discolouration.

Following investigations of osteoarthritis in man, several workers have considered that the subchondral bone plays an important role in the maintenance of the overlying cartilage (Sokoloff, 1963; Radin, Parker, Pugh, Steinberg, Paul and Rose, 1973; Pugh, Radin and Rose, 1974 and others). Radin et al. (1973) considered that

subchondral bone acted as a shock absorber, and that cartilage degeneration in osteoarthritis was associated with, and followed, remodelling of the subchondral bone in response to impact loading.

It is possible therefore, that remodelling of the subchondral bone plate of the fibrocartilage surface of the navicular bone in navicular disease cases, and, the vascular changes in the subchondral bone found on microangiography, may be responsible for and precede, degenerative changes in the fibrocartilage of the navicular bone.

The overall increase in vascularisation found in the navicular bones of the navicular disease group, and the absence of obvious vascular occlusion, suggests that a hyperaemic, rather than an ischaemic, factor is involved in the pathogenesis of navicular disease.

CHAPTER 4

PART II

FLUORESCENCE MICROSCOPY

LIGHT MICROSCOPY

FLUORESCENCE MICROSCOPY

INTRODUCTION

Examination of the navicular bones of a number of horses from the control and navicular disease groups was carried out following the administration of one or more intra-vital fluorochrome bone label.

A fluorochrome bone label is a substance which, following administration, is deposited at sites of mineralising tissue, for example in areas of new bone growth, and these labelled sites fluoresce when exposed to ultra-violet light.

The most commonly used of the intra-vital fluorochrome bone labels are the tetracyclines. The first report of their use as bone labels was by Milch, Rall and Tobie in 1958. Since then the tetracyclines have been used as fluorochrome bone labels by numerous workers, both in vivo (Harris, 1960; Holmes, 1963; Harris, Haywood, Lavorgna and Hamblen, 1968; Frost, 1969; and others), and in vitro (Tapp, Kovacs and Carrol, 1965; MacCallum, Latshaw and Kelly, 1971).

The mechanism by which tetracyclines are bound to the sites of new bone growth is not fully understood, but it is thought that they form complexes with calcium salts (Ibsen and Urist, 1964). It is considered that once a bone label is bound to bone, it will remain there until the bone is resorbed (Gilbertson, 1975).

Other intra-vital fluorochrome bone labels include, xylenol orange, alizarin red, calcein and fluorescein. Each of these substances will, when exposed to ultra-violet light, emit a different colour of fluorescence.

The use of more than one of these fluorochrome bone labels was advocated, because of this colour difference (Suzuki and Mathews,

1966; Olerud and Lorenzi, 1970; Rahn and Perren, 1971). to enable the sequence and rate of bone remodelling or development to be ascertained.

Other workers utilised the technique of multiple labelling with one type of tetracycline (Frost, Villanueva and Roth, 1960; Holmes, 1963; Melsen and Mosekilde, 1978), and relied on slight changes in the intensity of the yellow fluorescence to distinguish between the times of labelling.

Multiple labelling with different types of tetracyclines was carried out by Harris (1960), and Harris et al. (1968), and they maintained that the different tetracyclines could be distinguished by slight variations in the colour of their fluorescence.

Sequential fluorochrome bone labelling techniques can therefore afford a good dynamic picture of the sequence of events during new bone growth and bone remodelling.

MATERIALS AND METHODS

Fluorochrome bone labels were administered to 10 horses from the control group and 10 horses from the navicular disease group. Using the students' 't' test it was established that there was no significant difference in age between the two groups of horses. ($t = 1.61$; d.f. = 18; $P < 0.5$)

In most cases the fluorochrome bone label used was oxytetracycline hydrochloride* in a 5% solution. This was used for single, double, or triple labelling at time intervals indicated in TABLE 4.4, 4.5. In all cases the oxytetracycline was administered at a dose rate of 20 mg/kg bodyweight in a single dose, given by slow

* Engemycin 5%, Mycofarm Ltd., Braintree, Essex, England.

intravenous injection via the jugular vein using a 13 gauge intravenous catheter[‡].

In 2 horses xylenol orange⁺ was used in the bone labelling sequence. The dye was prepared for administration by the method described by Gilbertson (1975) and Duff (1980). The xylenol orange, at a dose rate of 90mg/kg bodyweight, as a 3% solution in a 2% solution of sodium bicarbonate[#], was administered by slow intravenous injection via the jugular vein, using a 13 gauge intravenous catheter.

All of the horses used in this study were kept under similar conditions, they were housed in loose boxes and given daily exercise, either walked out, lunged or given free exercise in paddocks.

Five of the 10 control horses and 5 of the 10 horses with navicular disease, selected randomly, were given the oral anti-coagulant warfarin^{**}, daily, at dose rates from 0.018 mg/kg to 0.08 mg/kg bodyweight, according to the protocols described by Colles (1979a)^{***}. Warfarin was withheld on the days of fluoro-chrome bone label administration.

In each case the last bone label was administered 7 days before euthanasia.

* Vygon (U.K.) Ltd., Cirencester, England.

+ B.D.H. Chemicals Ltd., Poole, England.

Hopkin and Williams, Chadwell Heath, Essex, England.

** Marevan, Duncan Flockhart & Co. Ltd., London, England.

*** See Chapter 5.

At post-mortem all four limbs were removed and the navicular bones dissected from the feet. Following radiography of these navicular bones, some were stored at -20° Centigrade, in order to preserve fluorescence until they could be sectioned, while others were sectioned immediately for examination by fluorescence microscopy.

Undecalcified bone sections for examination by fluorescence microscopy were cut from the intact navicular bone with a micro-slice 2 precision saw* using the technique described by Gilbertson (1975) and Duff (1980).

The microslice cuts by means of an annular blade with an electrometallic diamond cutting surface on its inner edge. The thickness of the sections cut can be controlled by moving the annular blade in amounts of 0.01 mm forwards or backwards.

The specimen to be sectioned is mounted, by means of dental impression wax⁺ of low melting point, onto a glass slide 3 mm thick which is itself mounted, by means of the same wax, into a table at one end of a counter balance pivot arm. The techniques used to mount these specimens are those described by Duff (1980).

Sections were cut parallel to the distal border of the bone or in the sagittal plane.

It was found, because of the extreme fragility of the internal trabecular framework of the navicular bone, that slow rotation speeds and frequent renewal of the annular blade were required to avoid damaging the section. Despite this it was found that sections

* Metals Research, Royston, Herts., England.

+ Kerr Impression Compound, Type 1, Kerr Sybron Corp., Michigan, U.S.A.

of the navicular bone could not be cut thinner than 100 μ ., and in most cases sections were between 100 μ and 150 μ in thickness.

In each navicular bone, two or three serial, 100 μ -150 μ sections were cut, for examination by fluorescence microscopy, then a 3 mm section was cut for histological examination, followed by another two or three serial 100 μ -150 μ sections for fluorescence microscopy.

The 100 μ -150 μ sections of navicular bone were then examined under the fluorescent microscope, or where this was not possible immediately following cutting, the sections were mounted between glass slides and stored at -20° centigrade.

The sections, before examination under ultra-violet light, were mounted in physiological saline on glass slides.

The fluorescent microscope used for this work was a Leitz* Orthoplan microscope with a Ploemopak 2.1., fluorescence vertical illuminator for incident-light excitation. For examination of oxytetracycline labelled bone, which emits a yellow fluorescence with a mean wave length of 440 nm (Koch, 1972), a filter system with, exciter filters KP 480 and KP 500, beam splitting mirror KP 510, and suppression filter K 515 was used.

An initial experiment was carried out to establish whether the administration of warfarin would affect the uptake of oxytetracycline by the bone. For this, 2 ponies aged 18 months old were used. Pony A received warfarin daily for 4 weeks prior to and 4 weeks post administration of oxytetracycline. Pony B received no warfarin prior to and post administration of oxytetracycline.

* Leitz (Instruments) Ltd., Luton, England.

	Pony A	Pony B
Bodyweight (kg)	183	165
Amount of warfarin administered per day at a dose rate of 0.018 mg/kg b.wt.:	3 mg	-
Amount of oxytetracycline hydrochloride (5%) administered in a single dose at a dose rate of 20 mg/kg b.wt.	73 mls	66 mls

At post-mortem the limbs were removed and prepared in the manner described previously.

RESULTS

Fluorochrome bone label was found to have been deposited in all of the navicular bones examined in this study.

The oxytetracycline fluoresced in bands of distinct, intense yellow colour (FIG 4.27). In the navicular bones of the horses in which oxytetracycline was used for double or triple labelling, the different times of labelling could be distinguished by the difference in intensity of yellow fluorescence between the bands, the earlier labelled areas fluorescing a less intense yellow.

The xylenol orange, where used, fluoresced an orange colour which was much less intense than the fluorescence of oxytetracycline (FIG 4.27). Although the xylenol orange labelled areas were easily distinguished from the oxytetracycline labelled areas, it was found that the xylenol orange faded if exposed to ultra-violet light for long periods.

Results of the administration of warfarin on the uptake of oxytetracycline by the navicular bones

On examination with the fluorescent microscope, no difference could be detected in the uptake and persistence of oxytetracycline between the navicular bone sections from the pony that had received warfarin, and the pony that had not received warfarin.

In the navicular bones of both ponies, areas of labelled bone were found throughout the navicular bone. The trabeculae and the subchondral bone of the fibrocartilage and articular surfaces showed areas of fluorescence and therefore new bone growth or bone remodelling. These labelled areas were interspersed with areas in which there was no uptake of bone label.

Results of fluorochrome bone labelling in 5 control horses not receiving warfarin treatment

Four of these horses received only oxytetracycline hydrochloride in the bone labelling sequence, and one horse received oxytetracycline hydrochloride and xylenol orange as the labels in a double labelling sequence. The doses and times of administration of these labels are contained in TABLE 4.4.

The navicular bones of all 5 horses showed areas of fluorescent bone label uptake. In the navicular bones of the 2 horses given a single bone label, single bands of yellow fluorescence were found. Distinct double bands of yellow fluorescence and some single bands of yellow fluorescence were found in the navicular bones of the horses double labelled with oxytetracycline, while bands of yellow and orange fluorescence were found in the navicular bones of the horse receiving oxytetracycline and xylenol orange.

In the navicular bones of the horse triple labelled with oxytetracycline, 3 bands of yellow fluorescence could just be distinguished in some of the areas of bone label deposition, but in other areas only double or single bands were found.

Areas of bone label deposition were found in all parts of the navicular bone.

Areas of both trabecular bone and the subchondral bone of the fibrocartilage and articular cartilage surfaces were labelled. Bone labelling was particularly evident in the bone immediately under the cartilages and around the trabeculae of the more distal parts of the navicular bone (FIGS 4.28 and 4.29).

No difference could be detected in the extent and distribution of the bone labelling between the fore and hind navicular bones.

The distance between the bands in the navicular bones from double and triple labelled horses varied in different parts of a section, and in different parts of the bone, and often only a single band of fluorescence was evident.

Results of fluorochrome bone labelling in 5 control horses receiving warfarin treatment

A similar bone labelling procedure was used in this group, as in the previous group not receiving warfarin treatment. The doses and times of administration of these bone labels, and the dose of warfarin required to produce an increase in prothrombin time of 2-4 seconds in each horse, are contained in TABLE 4.4.

No difference was found in the extent and distribution of fluorescent bone label in the navicular bones of these horses as compared to the navicular bones of the other 5 control horses.

Results of fluorochrome bone labelling in 5 horses with navicular disease, not receiving warfarin treatment

All of these horses received only oxytetracycline hydrochloride as the fluorochrome bone label, and the doses and times of administration are given in TABLE 4.5.

The navicular bones of all 5 horses showed areas of fluorescent bone labelling. In the diseased navicular bones of the horses triple labelled with oxytetracycline, 3 distinct bands of yellow fluorescence could be distinguished in some areas, while in others numerous bands of yellow fluorescence were found arranged in a more random manner and it was difficult to distinguish between the bands (FIG 4.30). In other areas of the triple labelled navicular bones double and single bands of yellow fluorescence were evident.

The extent of oxytetracycline uptake in the navicular bones of these horses with navicular disease was much greater than in either of the groups of control horses. A greater degree of bone label uptake was found in all parts of the navicular bone, but particularly by the trabeculae in the distal and central parts of the bone (FIG 4.31) and also by the subchondral bone of the fibrocartilage surface (FIG 4.32). Although in some sections there appeared to be an increase in bone label uptake by the subchondral bone of the articular surface, this was by no means as marked as the increase in uptake by the subchondral bone of the fibrocartilage surface. In all of the navicular bones with navicular disease, in the subchondral bone of the fibrocartilage surface, there was an overall increase in the number of bone labelled areas and this was most noticeable in the region of the central ridge.

As in the navicular bones of the labelled control horses, the

distance between the fluorescent bands in the navicular bones of the double and triple labelled horses from the navicular disease group varied throughout the navicular bone, and no particular pattern could be established. The distance between the bands of fluorescence in some areas appeared to be greater in the navicular bones of the navicular disease case than in the navicular bones of the control horse, although this was not assessed quantitatively.

All 5 of these horses showed no improvement in lameness during the period of study.

Results of fluorochrome bone labelling in 5 horses with navicular disease receiving warfarin treatment

All of these horses received only oxytetracycline hydrochloride as the fluorochrome bone label. The doses and times of administration of the oxytetracycline, and the dose of warfarin required to produce an increase in prothrombin time of 2-4 seconds in each horse, are given in TABLE 4.5.

No difference could be detected, in the extent and distribution of oxytetracycline labelled bone, in the navicular bones of these warfarin treated horses as compared to the navicular bones of the 5 horses with navicular disease not treated with warfarin.

The navicular bones of these warfarin treated navicular disease cases also showed a marked increase in uptake of the bone label as compared to the navicular bones of both groups of control horses.

All 5 of these warfarin treated navicular disease cases showed no improvement in their lameness during the period of study.

DISCUSSION

Bone labelling is a method whereby bone growth, and bone remodelling may be studied using an intra-vital stain. The bone label becomes deposited at sites of new bone formation and remains there unless removed by bone resorption.

The first report of bone labelling was by Payton in 1932 in pigs fed madder root, the active ingredient of which is alizarine. Alizarine itself was used by Schlour in 1936 to measure bone growth, but alizarine was subsequently found to have a high degree of systemic toxicity. Following the reports of Milch et al. (1958) on the use of tetracycline antibiotics as fluorochrome labels, "as effective histological indicators of newly proliferated bone tissue", bone labelling techniques became widely used, and several different fluorochrome bone labels were introduced, many of unknown toxicity.

Fluorochrome bone labelling has been used in the horse for examination of post natal ossification sites (MacCallum, Latshaw and Kelly, 1971) and to study growth rates in young horses (Goyal, MacCallum, Brown and Delack, 1981). Recently, Svalastoga, Reimann and Nielsen (1983) investigated the use of fluorochrome bone labelling in 5 young horses as a quantitative method of assessing bone formation in the navicular bones, using double labelling techniques with calcein and tetracycline.

All of these studies have involved young normal growing horses, and therefore the bone activity demonstrated by the uptake and deposition of the bone label will be age dependant and related to normal bone growth patterns.

The first report of an intra-vital bone label being used to

study navicular disease was by Østblum et al. (1982). They reported on the results of double labelling with oxytetracycline in 2 horses with navicular disease, but did not report on results of bone labelling in any control horses.

This present study has tried to examine bone remodelling and new bone growth in the navicular bones of adult control horses in order to establish the normal bone activity, if any, present in the navicular bone. This was necessary before any comparisons could be made with bone activity in navicular bones from navicular disease cases.

For these purposes 10 control horses and 10 horses with navicular disease were used. Since age is an important factor in bone activity, and since Branscheid (1977) established that the navicular bone development was not complete until approximately 18 months old, it was important that no horse less than 2 years old should be included in the bone labelling study. It was important also that there should be no significant difference in age between the 2 groups of horses. Five of these control horses and five of the horses with navicular disease, randomly selected, were given warfarin treatment (Colles, 1979a and 1982) in order to examine the possible effects of this drug on bone remodelling.

Before this could be carried out it was first necessary to establish whether the administration of warfarin would affect the uptake and persistence of the bone label. Young ponies were used for this experiment since it was known that their navicular bones were still developing and were therefore likely to show substantial uptake of fluorochrome bone label.

No difference was found in the extent and distribution of the

oxytetracycline labelled areas in the navicular bones of these 2 ponies. It was concluded from this experiment that the administration of warfarin was unlikely to affect the uptake and persistence of oxytetracycline by the navicular bone.

The results of the fluorescent bone labelling in these ponies are in agreement with those of Svalastoga et al. (1983).

It was decided early in this study to use oxytetracycline as the sole fluorochrome bone label, and rely on the change in intensity of the yellow fluorescence to distinguish between the times of labelling.

Although the fluorochrome bone label xylenol orange was used in the two horses as the last of 2 labels, it was considered that the results of double and triple labelling with oxytetracycline were as good as those with oxytetracycline and xylenol orange. From these results, plus the high cost of xylenol orange and its unknown toxicity in the horse it was decided to use oxytetracycline on its own. The oxytetracycline was administered at a dose rate of 20 mg/kg body weight, although bone labelling with oxytetracycline has been achieved at lower dose rates (Goyal et al., 1981; Svalastoga et al., 1983).

Despite the report of Andersson, Ekman, Mansson, Persson Rubarch and Tufvesson (1971) of lethal complications following the administration of large doses of oxytetracycline in horses, slight adverse reaction was shown by only 2 horses, one from the control group (A65), and one from the navicular disease group (B60). The adverse reaction was expressed in the form of sweating, muscle tremors, and signs of mild colic, which lasted for 3 - 4 hours following injection of the oxytetracycline. In both cases no further bone labelling was carried out.

The results of fluorochrome bone labelling in the 5 control horses not receiving warfarin treatment indicate that bone remodelling is a feature of the normal navicular bone. Areas of bone label uptake and therefore bone deposition were found throughout the navicular bone, both around the trabeculae and in the subchondral bone of the fibrocartilage and articular surfaces, particularly in the region immediately beneath the cartilages.

Bone remodelling is a normal feature of bone in all animals, throughout life, in response to both metabolic and mechanical factors. It has been suggested that bone remodelling processes may be controlled in some way by customary intermittent deformation of bone, and that this repeated deformation to some extent may be a normal requirement rather than entirely destructive (Lanyon, 1982).

Radin et al. (1973) suggested that limited trabecular micro-fracture in subchondral bone was part of the shock absorbing mechanism and in normal circumstances there would be a balance between fracture and fracture healing.

The navicular bone under normal circumstances because of its anatomical position is subject to a great deal of pressure associated with weight bearing.

These factors may account for the uptake and distribution of bone label displayed by normal navicular bones.

No quantitative analysis of the rate of bone remodelling such as that described by Melsen and Mosekilde (1978) was carried out in this study, since it was considered that too many variables were present.

The results of fluorochrome bone labelling in the 5 horses with navicular disease showed that in every case the extent of bone label

uptake by the affected navicular bones was markedly increased in comparison to the uptake by the navicular bones of the control horses. Although quantitative analysis was not carried out, the differences between the navicular bones of the two groups were obvious.

This increase in oxytetracycline uptake appeared to involve areas of bone in all parts of the navicular bone, but it was particularly marked around the trabeculae in the central and distal parts of the bone, and in the subchondral bone of the fibrocartilage.

These findings are consistent with the radiographic changes in trabecular pattern and subchondral bone of the fibrocartilage surface of the navicular bones from navicular disease cases, noted earlier in this chapter.

These findings were also consistent with those of Østblum et al. (1982), who considered that there was a high rate of bone turnover present in all parts of the navicular bone in navicular disease cases.

No difference in the extent and distribution of bone labelled areas was noted between the navicular bones of the 5 control horses receiving warfarin treatment, and the 5 control horses not receiving warfarin treatment.

It would therefore seem likely from these results that warfarin has little or no affect on the normal processes of bone remodelling.

Furthermore, no difference was noted in the extent and distribution of bone labelled areas between the affected navicular bones of the 5 horses with navicular disease receiving warfarin treatment, and the 5 horses with navicular disease not receiving warfarin treatment. However, these results are inconclusive since

none of the 5 horses with navicular disease receiving warfarin treatment had shown any response to the warfarin treatment.

Histological examination of sections of these bone labelled navicular bones and other navicular bones from control horses and horses with navicular disease was carried out to confirm and elucidate these findings.

LIGHT MICROSCOPY

Following gross pathological examination and radiography of the navicular bones from control horses and horses with navicular disease, histological examination of sections of these navicular bones was carried out.

The fore navicular bones from 22 control horses and the adjacent portion of the deep flexor tendons from 10 of these control horses were examined histologically. The fore navicular bones and adjacent portion of the deep flexor tendons from 25 horses with navicular disease were examined histologically. In addition histological examination was carried out on the hind navicular bones from 8 control horses and 3 horses with hind limb navicular disease.

MATERIALS AND METHODS

The navicular bones and deep flexor tendons, either whole or sectioned as in FIG 4.12, were placed in 10% buffered formalin for 7-14 days for complete fixation.

The navicular bones were then transferred into Gooding Stewart's solution, for decalcification. The decalcifying fluid was changed at frequent intervals, and the navicular bones or navicular bone sections were radiographed at 1-2 week intervals until decalcification was complete. This process required from 6 to 20 weeks depending on the size of the specimen. On completion of decalcification, the navicular bones or navicular bone sections were dehydrated through alcohol and embedded in paraffin wax.

The 3 mm sections from the navicular bones of fluorescent bone labelled horses were prepared for histological examination in the same manner. Following fixation the deep flexor tendons were also

dehydrated through alcohol and embedded in paraffin wax.

Routinely histological sections were cut parallel to the distal border 2-3 mm into the navicular bone as recommended by Colles (1982) and/or in the sagittal plane. These sections were stained routinely with haematoxylin and eosin, and Van Gieson. Histological sections were cut from the deep flexor tendons in the same planes as the navicular bone sections, which were either at right angles to the tendon fibres or parallel to the tendon fibres.

RESULTS

The results of histological examination are described in relation to the grades of gross pathological changes recorded earlier in this chapter.

No visible lesion on gross pathological examination of the navicular bone

This finding was recorded only in the navicular bones of control horses on gross pathological examination. Histological examination of the navicular bones of these horses showed that the posterior surface of the navicular bone in contact with the deep flexor tendon, was covered with normal fibrocartilage of equal thickness over the whole surface, while the anterior surface of the navicular bone, which articulates with the second and third phalanges was covered with normal hyaline cartilage. The hyaline cartilage of the articular surface was thicker than the fibrocartilage of the fibrocartilage surface. No histological lesions were found in the articular cartilage or fibrocartilage of these navicular bones.

Beneath the cartilage layers were subchondral bone plates composed of lamellar bone. Concentric lamellae of Haversian

systems and interstitial lamellae could be identified in these subchondral bone plates. In all cases the subchondral bone plate of the fibrocartilage surface was thicker than that of the articular surface, particularly in the region of the central ridge. Blood vessels were found in the Haversian canals (FIGS 4.33 and 4.34).

The medulla of the navicular bone was composed of a fine, organised, network of trabecular bone. Between the trabeculae there was a fine connective tissue network in which blood vessels and numerous fat cells were evident. Blood vessels were more numerous in the distal parts of the navicular bone (FIG 4.35).

Around the edges of a small number of trabeculae and around some of the vascular channels in the subchondral bone plates, particularly in the area of bone immediately below the calcified layer of cartilage, active osteoblasts were identified. Osteoclasts were occasionally noted in similar areas, but they were present to a lesser extent.

Slight orange/brown discolouration and slight erosion of the fibrocartilage of the navicular bone

This gross pathological lesion was found in the navicular bones of control horses and in a small number of navicular bones of navicular disease cases.

On histological examination of these navicular bones from both control horses and horses with navicular disease, slight thinning and roughening of the surface of the fibrocartilage was evident mostly in the region of the central ridge, the rest of the fibrocartilage appearing normal.

A slight increase was noted in the number of blood vessels in

the inter-trabecular spaces, particularly in the more distal parts of the bone, and to a lesser extent in the subchondral bone of the fibrocartilage surface. These changes were more noticeable in the navicular bones of the navicular disease cases than in the navicular bones of the control cases. In the subchondral bone plate of the fibrocartilage surface in the navicular disease cases, more osteoclasts and osteoblasts were evident than in the normal navicular bone. A similar increase was not noted in the subchondral bone plate of the articular surface.

Distinct yellow discolouration and erosion of the fibrocartilage surface of the navicular bone

This gross pathological lesion was present in only a small percentage of navicular bones in the control group, and in a large percentage of the navicular bones in the navicular disease group.

In the navicular bones of both the control cases and the navicular disease cases histologically there was thinning of the fibrocartilage, particularly in the region of the central ridge. In some cases proliferation of cartilage cells, cell clumps and slight fibrillation was evident around the fibrocartilage lesions.

In the subchondral bone plate beneath the fibrocartilage lesions there was an increase in the number of blood vessels and fibrous connective tissue present and a decrease in the amount of subchondral bone. This change was more marked in the navicular disease cases than in the control cases. There was also an increase in the number of blood vessels and fibrous connective tissue in the inter-trabecular spaces. In most of the navicular bones in the navicular disease cases, more osteoblasts were present around

the trabeculae, and new bone was evident over the surface of some of these trabeculae (FIG 4.36).

Distinct yellow discolouration and marked erosion of the fibrocartilage surface of the navicular bone

This degree of gross pathological change was found only in the navicular bones of the navicular disease cases.

Histologically there was marked thinning of the fibrocartilage, particularly over the central ridge, and proliferation of cartilage and fibrillation of the cartilage around the edges of these lesions. In some cases, in areas around the central ridge, there was complete loss of fibrocartilage exposing small areas of subchondral bone. An increase in the amount of vascular granulation tissue and a decrease in the amount of subchondral bone was found in the subchondral bone plate of the fibrocartilage surface. Increased osteoclastic activity was evident particularly in the region immediately below the fibrocartilage. In this area also, small projections of vascular granulation tissue were noted from the subchondral bone into the fibrocartilage (FIG 4.37).

Osteoblasts were also present in the subchondral bone of the fibrocartilage surface, but these were most in evidence around trabeculae particularly in the central and distal parts of the navicular bone.

Similar changes were noted in the subchondral bone plate of the articular surface, but to a much lesser extent than in the subchondral bone of the fibrocartilage surface.

Red/brown discolouration and marked erosion of the fibrocartilage to expose the underlying bone

This degree of gross pathological change was only found in navicular bones from navicular disease cases.

Histologically in most of the navicular bones examined the fibrocartilage was absent in areas around the central ridge, and the rest of the fibrocartilage was thinned, except towards the edges of the navicular bone, where there was some proliferation of cartilage cells. At the base of the fibrocartilage erosion there was only a very thin plate of bone, or in some cases small areas of subchondral bone were absent, and the base of the erosion was lined with vascular granulation tissue (FIG 4.38).

The amount of subchondral bone beneath the fibrocartilage was greatly reduced and this was replaced by vascular and fibrous granulation tissue. In some areas this vascular granulation tissue projected from the subchondral region into the fibrocartilage.

An increase in the number of osteoblasts was found around the trabeculae in the medulla of the bone. The new bone production by these osteoblasts had resulted in the thickening of the trabeculae and loss of their normal organised appearance.

Red/brown discolouration and severe erosion of the fibrocartilage to expose the underlying bone in which small cavities were evident

With this degree of gross pathological change in the navicular bone, histologically the fibrocartilage changes were similar to the previous grade of gross pathological change, except that there was a greater loss of subchondral bone. The areas of subchondral bone resorbed by osteoclasts were replaced by granulation tissue (FIG 4.39).

Complete loss of fibrocartilage and collapse of the thinned bone plate was evident in areas of the fibrocartilage surface, particularly in the region of the central ridge. Around the edges of these lesions islands of bones were found surrounded by osteoclasts. In some cases the osteoclastic activity in the subchondral bone of the fibrocartilage had extended to involve areas of trabecular bone immediately beneath it.

Marked trabecular thickening and loss of their organised appearance, was present in the medulla of the navicular bone around these lesions (FIG 4.40).

Red/brown discolouration and severe erosion of the fibrocartilage exposing the underlying bone in which cavities were present with adhesions of the deep flexor tendon to the navicular bone

The histological appearance of the navicular bones showing this degree of gross pathological change is similar to the navicular bones with the previous grade of gross pathological change, except that areas of collapse of the subchondral bone plate is greater.

The defect in the subchondral bone was filled with fibrous granulation tissue, to which was attached through the space in the subchondral bone, tendon fibres from the deep flexor tendon (FIG 4.41).

In some cases where fibrocartilage remained, there was proliferation of cartilage cells at the edges of the cavity.

The hind navicular bones from the control horses showed the same type of histological picture as the fore navicular bones from the control horses. The affected hind navicular bones from horses with hind navicular disease showed histological changes

similar to the fore navicular bones from the horses with navicular disease with the same degree of gross pathological change.

The navicular bone sections from the fluorescent bone labelled horses confirmed the findings of the previous section. The areas of bone label uptake were, histologically, the areas in which active osteoblasts were producing new bone, namely around the trabeculae and in the subchondral bone of the fibrocartilage surface.

Histological examination of the deep flexor tendon

In the control horses, sections from the deep flexor tendon in which there was no visible lesion, or only faint orange/brown discolouration on gross pathological examination, when examined histologically had all the appearance of a normal tendon as described by Bloom and Fawcett (1968).

This histological picture was present in all of the deep flexor tendons examined from the control horses and in 4(8%) of the tendons from the navicular disease cases examined (FIG 4.42).

Deep flexor tendons with yellow discolouration and slight roughening of the superficial fibres was found in a small percentage of horses from the control group and a large percentage of the horses from the navicular disease group. The histological changes were the same in both groups, there was disruption of the tendon fibres and loss of some fibroblasts, these changes being confined to the superficial layers of the tendon.

As the degree of gross pathological change found in the deep flexor tendon increased, histologically the degree of tendon fibre disruption and loss of fibroblasts increased. These histological changes were not just confined to the superficial layers of the

tendon but extended a short way into the body of the tendon. In some cases in which there was severe disruption and necrosis of tendon fibres, the whole tendon appeared thinned on cross-section.

Occasionally cartilage cells were evident between some of the disrupted tendon fibres.

No evidence of an inflammatory reaction nor any evidence of reparative processes were found in any of the deep flexor tendons examined (FIG 4.43).

DISCUSSION

The histological appearance of the normal navicular bone and the navicular bone from navicular disease cases, was first described from undecalcified bone sections, by Smith in 1886. Since then numerous workers have described the histopathological appearance of the navicular bones from navicular disease cases (Wilkinson, 1953; Wintzer, 1964; O'Brien et al., 1975; Colles, 1979 and 1982; Østblum et al., 1982).

Unlike histological examinations of periodic biopsy samples, histological examination of the navicular bone, as with other forms of pathological examination of the navicular bone, is restricted because the pathological changes found are only the changes present at the time of post-mortem. The processes by which these changes developed, and the way in which they may progress, cannot be ascertained. It is only by histological examination of a number of navicular bones from navicular disease cases at different stages in the disease, that the course of the pathological processes may be determined.

From gross pathological examination of the navicular bones

and deep flexor tendons from the horses in this study, it was evident that different degrees of pathological change were present on the fibrocartilage surface of the navicular bone and the contiguous surface of the deep flexor tendon. These different degrees of gross pathological change could be fitted into a pattern of increasing severity.

When the results of histological examination of these navicular bones were related to the degree of gross pathological change, in each case it was found that the degree of histological change increased as the degree of gross pathological change increased. Histologically changes were most evident in the fibrocartilage and underlying subchondral bone plate of the fibrocartilage surface of the navicular bone, and around the trabeculae and in the inter-trabecular spaces in the medulla of the navicular bone.

As the degree of gross pathological change on the fibrocartilage surface of the navicular bone increased, histologically it was found that:

- (i) The amount of fibrocartilage decreased, particularly in the region of the central ridge, while in other areas there was cartilage cell proliferation, cell clumping and fibrillation.
- (ii) The disruption, and loss of the subchondral bone plate of the fibrocartilage surface increased.
- (iii) The overall vascularisation of the navicular bone increased.
- (iv) The thickness of the trabeculae in the medulla of the bone increased and they became increasingly disorganised.
- (v) The osteoblastic and osteoclastic activity in the navicular bone increased.

Some of the navicular bones of the control cases also had a

degree of gross pathological change in the cartilage of the fibrocartilage surface. Histologically these navicular bones differed slightly from the navicular bones in which no visible lesions were found in that they showed a slight increase in vascularisation.

In the small number of control navicular bones in which a distinct yellow discolouration and erosion was found on the fibrocartilage, a slight degree of disruption of the subchondral bone plate of the fibrocartilage surface was evident, but this was much less marked than in the navicular bones from the navicular disease cases with the same degree of gross pathological change.

The histological changes found in these navicular bones from control cases may arise because:

- (i) These cases are early navicular disease cases in which no clinical signs are evident.

or

- (ii) A degree of subchondral bone remodelling is "physiologically tolerable" (Radin et al., 1973).

As the degree of gross pathological change on the surface of the deep flexor tendon increased, histologically there was an increase in disruption of tendon fibres and loss of fibroblasts in the surface layers of the deep flexor tendon. Very little attempt at repair was evident on the part of the tendon. These results confirm the findings of Wilkinson (1953).

In some cases cartilage cells were evident in the superficial layers of the damaged tendon. Smith (1886) and Wilkinson (1953) noted cartilage type cells in the deep flexor tendon from navicular disease cases. While Smith (1886) considered these to be derived from the damaged fibrocartilage of the navicular bone, Wilkinson

(1953) considered that they were derived from the peritendinium of the deep flexor tendon. The latter situation is more likely since cartilage cells are normally present in tendons at specific sites such as at their insertions to bone.

The results of histological examination in this study of the navicular bones from navicular disease cases confirm the findings of Wilkinson (1953), Wintzer (1964), O'Brien et al. (1975) and Østblum et al. (1982). All of these workers reported an increased vascularisation of the navicular bone, and histological changes affecting the subchondral bone of the fibrocartilage surface early in the course of navicular disease. Wintzer (1964) maintained that the pathological changes in the fibrocartilage followed "subchondral bone atrophy".

In this present study, subchondral bone loss in the fibrocartilage surface of the navicular bone was found in all of the navicular bones from the navicular disease cases, even in cases in which only slight gross pathological change was evident in the fibrocartilage..

Numerous workers have maintained that the subchondral bone has an important role in the maintenance of the overlying cartilage. (Sokoloff, 1963; Radin et al., 1973; and others). Sokoloff (1963) reported that cartilage deteriorated where it come in contact with blood vessels, as can occur in osteoarthritis. In this present study projection of vascular granulation tissue into the base of the fibrocartilage was noted in many of the navicular bones from navicular disease cases.

Colles (1979 and 1982) maintained from histological examination of navicular bones from navicular disease cases that,

in no case were gross pathological lesions present in the absence of arterial thrombosis of arteries within the navicular bone, and that areas of bone necrosis in these navicular bones were the result of localised ischaemia.

In this present study no evidence of bone necrosis was found in any of the navicular bones examined. However, arterial thrombosis affecting a small number of larger arteries was found in 5 (11%) navicular bones from the navicular disease cases examined histologically and in 3 navicular bones from the control horses examined histologically, but in no case were these associated with areas of bone necrosis. The navicular disease cases in which arterial thrombii were found all had severe pathological changes present in the navicular bones, and it is unlikely that the small number of affected arteries could have resulted in these changes. As Colles (1982) pointed out, a degree of arterial thrombosis may be a normal continuous process in some adult horses.

The results of histological examination of the navicular bones in this study confirm the findings of radiography, microangiography, and fluorescent bone labelling discussed earlier in this chapter.

It is considered from histological examination of the navicular bones from control horses and horses with navicular disease that increased vascularisation, increased bone remodelling and degeneration of fibrocartilage are the major histological changes present in navicular disease.

CHAPTER 4.

PART III

GENERAL DISCUSSION AND CONCLUSIONS

GENERAL DISCUSSION AND CONCLUSIONS

It has been suggested that navicular disease is not a specific entity, but the result of different pathological processes affecting the navicular bone (Hickman, 1964).

In this study the diagnosis of navicular disease was based entirely on clinical criteria, so that the radiographic and pathological examinations could be carried out to assess whether the clinical signs were the result of the same or different pathological processes.

As navicular disease has not yet been produced experimentally in the horse, all the material for pathological examination was from naturally occurring cases.

Although in the pathological examination of navicular bones and deep flexor tendons, of a number of navicular disease cases, different stages of the disease process will be found, it is difficult to assess which are primary pathological lesions and which are secondary.

In this study pathological examinations were also carried out on the navicular bones and deep flexor tendon of control horses. Since joints are the subject of constant pressures and stresses, it was considered particularly important to examine control horses in order to establish the extent of "normal wear and tear" changes.

All of the affected navicular bones of the navicular disease cases had pathological changes in the fibrocartilage, and subchondral bone of the fibrocartilage surface. The articular cartilage and subchondral bone of the articular cartilage was not affected.

In most cases pathological changes were evident in the adjacent portion of the deep flexor tendon, but were always

accompanied by changes in the fibrocartilage surface of the navicular bone. It is considered that pathological changes in the deep flexor tendon in navicular disease are secondary to changes in the fibrocartilage surface of the navicular bone, and that adhesions to the navicular bone develop as part of the tendon healing process (McMinn, 1969).

Different degrees of pathological change, which could be fitted into a pattern of increasing severity, were found in the navicular bones and deep flexor tendons, in this study.

The main pathological changes found in all navicular disease cases, from gross pathological examination, radiography, microangiography, and fluorescent and light microscopy were:

- (i) Degenerative changes of the fibrocartilage of the navicular bone.
- (ii) Disruption and loss of the subchondral bone plate of the fibrocartilage surface of the navicular bone, particularly in the region of the central ridge.
- (iii) An overall increase in the vascularisation of the navicular bone.
- (iv) An increased rate of bone remodelling, particularly in the subchondral bone plate of the fibrocartilage surface.
- (v) An increased thickness and loss of organisation of the trabeculae in the medulla of the navicular bone.
- (vi) Disruption of tendon fibres of the deep flexor tendon.

Some of these pathological changes were found in a small number of control horses but these changes were less severe than in the navicular disease cases. It is possible that the degree of pathological change found in the control cases is associated with

"normal wear and tear" or that these cases were displaying early changes of navicular disease.

A significant positive correlation was found between the degree of gross pathological change present in the navicular bone and deep flexor tendon, and the navicular score as calculated from routine antero-posterior radiographs in all of the navicular bones examined. It is therefore considered that the navicular scoring system gives an indication of the degree of pathological change in navicular disease.

Since it is considered that an increase in number and change in shape of the distal nutrient foramina, upon which this scoring system is based, is an indication of an increased periosteal blood supply to the navicular bone (Colles, 1982a), it is likely that the navicular scoring system can also be a guide to the degree of vascularisation of the navicular bone.

Many workers have suggested that local vascular changes may be important in the pathogenesis of navicular disease, but opinions differ regarding the nature of these changes. Some workers have suggested that the vascular involvement in navicular disease was occlusive in nature (Nemeth, 1972; Colles and Hickman, 1977; Colles, 1982; Fricker et al., 1982) while others have maintained that hyperaemia of the navicular bone was involved in the development of navicular disease (Smith, 1886; Olsson, 1954; O'Brien et al., 1975).

Similarly, in osteoarthritis in man, some workers support the view that ischaemia of bone is important in the pathogenesis (Phemister, 1940; Pridie, 1952) while others consider that hyperaemia of the bone is involved (Harrison et al., 1953; Brookes and Helal, 1968; Arnoldi, Linderholm and Mussbrichler, 1972).

The findings of this present pathological study do not support the hypothesis that occlusive vascular disease and local ischaemia are involved in the development of navicular disease (Colles and Hickman, 1977; Colles, 1982). No evidence of bone necrosis was found, and although arterial thrombi were observed in a small number of navicular bones from navicular disease and control cases, these were not associated with bone necrosis.

The overall increase in vascularisation found in the navicular bones of navicular disease cases, in the absence of obvious vascular occlusion and of bone necrosis, suggest a hyperaemic rather than ischaemic factor may be involved in the development of navicular disease.

The cause of the hyperaemia in the navicular bones of navicular disease cases has not been established.

Congestion or hyperaemia may be produced either by the increase in the flow of blood to an area (arterial or active hyperaemia) or by interference to blood flow from an area (venous or passive hyperaemia) (Cappell, 1964).

Harrison et al. (1953) maintained that active hyperaemia of the subchondral bone occurred in osteoarthritis of the hip in man, in response to degeneration of the articular cartilage.

However, numerous workers considered that the subchondral bone plays an important role in the maintenance of the overlying cartilage, and that cartilage degeneration in osteoarthritis was associated with and followed changes in the subchondral bone. An increase in vascularisation was found in the subchondral bone plate of the fibrocartilage surface of navicular bones in the navicular disease cases examined in this study, and was reported from

microangiographic studies by Svalastoga (1983). It is suggested that the pathological changes found in the fibrocartilage of the navicular bone are associated with, and follow changes in the subchondral bone plate of the fibrocartilage surface of the navicular bone.

In contrast Brookes and Helal (1968) and Arnoldi et al. (1972) using intra-osseous phlebography and measuring intra-osseous pressure, considered that venous congestion (passive hyperaemia) was associated with osteoarthritis in man. Svalastoga and Smith (1983) have similarly shown an increase in subchondral bone pressure, and a lengthening in pressure drop time in the navicular bones of navicular disease cases, consistent with venous congestion. Colles (1982) showed, from angiographic studies of blood flow, that there was a marked slowing of circulation time through the foot in navicular disease cases.

~ It has been suggested that subchondral venous congestion is an important factor in the development of cartilage degeneration (Brookes and Helal, 1968) and that venous congestion stimulates structural changes in cancellous bone (Brookes and Helal, 1968; Arnoldi, Linderholm and Vinnerberg, 1972).

From these findings and the results of pathological examinations in this study, it is suggested that hyperaemia is involved in the pathogenesis of navicular disease and that this hyperaemia may be:

- (i) Arterial or active - secondary to mechanical damage or increased functional activity of the subchondral bone plate of the fibrocartilage in response to impact loading or altered mechanical stress. This may result in disruption and loss of subchondral bone, degeneration of the overlying

fibrocartilage and altered remodelling of trabecular bone.

or it may be:

- (ii) Venous or passive - associated with venous stasis of the foot.

The venous congestion in the navicular bone may be responsible for the degenerative changes observed in the fibrocartilage either as a primary factor or secondary to disruption of the subchondral bone plate, and for the increased bone remodelling found in the navicular bones in navicular disease cases.

or it may be:

- (iii) A combination of (i) and (ii).

It is concluded from gross pathological examination, radiographic and microangiographic examinations, fluorescent, and light microscopic examinations of navicular bones and deep flexor tendons of control horses and horses with clinical evidence of navicular disease that:

- (i) The affected navicular bones and deep flexor tendons, from all of the navicular disease cases examined, had pathological changes of different degrees, affecting the fibrocartilage and subchondral bone plate of the fibrocartilage surface of the navicular bone, the trabeculae in the medulla of the bone, and the contiguous portion of the deep flexor tendon. These pathological changes could be fitted into a pattern of increasing severity.
- (ii) Some of the navicular bones and deep flexor tendons from control horses similar but less severe pathological changes, which in some cases may be associated with "normal wear and tear".

- (iii) The articular surface of the navicular bone is not involved in navicular disease.
- (iv) The navicular scoring system is a useful indicator of the possible degree of pathological change present in the navicular bone and deep flexor tendon in navicular disease cases, but should not be used as certain evidence of these changes.
- (v) The overall increase in vascularisation and bone remodelling in the diseased navicular bone, in the absence of vascular occlusion and bone necrosis, suggests that hyperaemia rather than ischaemia is involved in the pathogenesis of navicular disease.

CHAPTER 5

THE TREATMENT OF NAVICULAR DISEASE

"If his 'oss is not so good as it might be let him cherish the reflection that it might have been far worse!

Mr. Jorrocks.

REVIEW OF THE LITERATURE

Over the past two centuries the treatments used for navicular disease have been many and varied. The majority of workers have stated that navicular disease was incurable and that most forms of treatment were palliative and aimed at prolonging the useful life of the horse (Fitzwygram, 1894; O'Connor, 1946; Hickman, 1964; Schebitz, 1964; Baum, 1974 and others). Drake (1896) considered that no cure could be found in "an established case of navicular disease if disorganisation has taken place". Others believed that if treatment was instigated early in the course of the disease, a cure could be affected in a few cases (Percivall, 1873; Reeks, 1925; Donahue, 1935). Likewise Østblum et al. (1982) proposed that navicular disease was reversible, and that it was only secondary changes such as adhesions between the navicular bone and deep flexor tendon, or spur formation on the navicular bone which rendered the condition irreversible.

The first form of treatment suggested for navicular disease was the passing of a seton through the frog, and if this was unsuccessful, then firing of the coronary band was indicated (Bridges, 1752). Other workers followed this course of treatment (Sewell, 1837, cited by Smith, 1930; Fitzwygram, 1894), the purpose being as O'Connor (1946) explained, to encourage adhesions between the navicular bone and the deep flexor tendon. By 1925 (Reeks) this practice appeared to have ceased.

Percivall (1873) who was not in favour of either frog setoning or firing of the coronary band, suggested two forms of treatment. In the early stages of the disease he advocated a "mild or soothing" treatment, which included immersion of the foot in warm water baths, bran poultices and purgatives. The treatments recommended for the

later stages of navicular disease were, "blood letting" and blistering of the pastern and coronary band. As a last resort in the incurably lame horse Percivall (1873) suggested neurectomy.

NEURECTOMY

Neurectomy was defined by Percivall (1873) as the "division of a nervous cord, and the subsequent excision of a portion of it, with the view of removing pain through the destruction of feeling".

Sewell in 1817 (cited by Smith, 1930) claimed to have discovered neurectomy as the treatment for navicular disease but Moorcroft (1819)* reported that he had performed the operation for the treatment of navicular disease some 18 years previous to this. This form of treatment was considered a last resort by a number of workers, (Fitzwygram, 1894; Reeks, 1925; Hickman, 1964; Johnson, 1982). O'Connor (1946), Schebitz (1964) and Adams (1974) on the other hand maintained that neurectomy was the only course of action likely to render the horse useful for a period. Neurectomy was not considered to be a cure for navicular disease, it relieved the pain but not the disease processes (Fitzwygram, 1894 and others). Leeney (1898) in contrast believed that neurectomy could actually cure navicular disease. Colles (1982) suggested that if similar sympathetic controls over blood vessels' size existed in the horse's foot as in the human finger then neurectomy could result in dilation of the blood vessels and so increased blood flow through the foot, and in this way possibly aid recovery from navicular disease.

Complications have arisen following neurectomy for navicular disease and these include; an increased tendency for the horse to stumble and fall, particularly if neurectomy had been carried out in both front feet; undetected wounds in the foot and heel region;

* Letter to editor of the Calcutta Journal, 1819, published in the Veterinarian, 1830.

painful neuromas developed on the end of the cut nerves; rupture of the deep flexor tendon; sloughing of all or part of the hoof wall as a result of occlusion of its blood supply; fracture of the navicular bone; regeneration of the nerves after 6 months or more; the presence of accessory nerve branches which may result in incomplete desensitisation (Moorcroft, 1819; Percivall, 1873; Reeks, 1925; Schebitz, 1964; Adams, 1974 and others). Schebitz (1964) reported that 13 horses (16%) out of a total of 81 horses he had treated for navicular disease by neurectomy had shown complications. Wintzer (1964) could find no significant difference between neurectomy and other forms of treatment for navicular disease.

CORRECTIVE SHOEING

The most widely reported treatment for navicular disease is corrective shoeing. Frost (cited by Donahue, 1935) suggested that affected horses should be shod so as to give them earlier breakover at the toe. He described the preparation of shoes with a bar which applied frog pressure well behind the navicular bone. The egg bar shoe recommended by Østblum et al. (1982), for which they reported a high degree of success in treating navicular disease cases, somewhat resembled that of Frost. In the case of early navicular disease, McCunn (1951) wrote, "much can be done with the help of a competent farrier". He advocated paring the foot at the toe as much as possible while leaving the heels long. He then applied a shoe with a rolled toe, narrow branches so that the pressure was on the wall of the foot, and wedged heels. The shoe removed all frog pressure and could result in immediate improvement in the animal's lameness.

Shoes with rolled toes and raised heels were also recommended for navicular disease cases by Hickman (1964), Moyer (1979), Adams (1974) and Johnson (1982). Adams (1974) in addition suggested the application of a bar across the middle third of the frog, to protect the frog from ground pressure, and a rubber pad under the shoe as a hoof cushion. Another method of raising the heels of the foot mentioned by Moyer (1979) was to use wedge nylon pads under a flat shoe. Colles (1982a) considered that shoes with raised heels or nylon wedges would help straighten the hoof/pastern axis but would not correct what he believed to be one of the underlying defects predisposing to navicular disease, namely low heels and long toe conformation. He advocated applying a wide shoe set well back at the toe with the branches of the shoe extending beyond the bearing surface of the heels. In addition to corrective shoeing, grooving of the hoof wall was recommended by McCunn (1951), Hickman (1964) and Adams (1974). This process was well described by Hickman (1964), and involved cutting a number of parallel grooves from the coronary band to the ground surface of the hoof wall of the inside and outside heel. These grooves were three quarters of an inch apart and extended down to the white line, the purpose being to relieve pressure at the heels.

REST

Hickman (1964) suggested that 3-4 months rest at grass could arrest the disease in some horses, and on this subject Reeks (1925) wrote;

The period of rest cannot be satisfactorily advised, and the practitioner is wise who makes it a long one. Rest should be advised, in fact, long after symptoms of lameness have disappeared and recovery is judged to have taken place.

Rest as a treatment for navicular disease had not produced satisfactory results according to Wintzer (1964). Johnson (1982), although he advocated rest and corrective shoeing for cases of navicular bursitis with no radiographic changes, also maintained that horses with navicular disease should be exercised daily.

ANALGESICS AND ANTI-INFLAMMATORY DRUGS

The use of analgesics such as phenylbutazone (Baum, 1974 and Johnson, 1982) and anti-inflammatory agents such as orgotein (Coffman, Johnson, Fritschler, Garnet and Scrutchfield, 1979) have been suggested to prolong the working life of horses affected with navicular disease. Their effect is to reduce the pain and so lameness rather than treating the condition itself (Colles, 1982a). Baum (1974) pointed out that with progress of the disease, the effect of this form of therapy was reduced. Ackerman et al. (1977) could find no advantage for analgesics combined with corrective shoeing, over neurectomy for the treatment of navicular disease. Johnson (1982) considered that analgesic drugs should not be administered continuously to a horse with navicular disease if the horse was not to be ridden consistently, but administered in relation to exercise.

CORTISONE INJECTION INTO THE NAVICULAR BURSA

The injection of cortisone into the navicular bursa as a therapy for navicular disease was reported by Wilkins (1957). He would appear to have injected cortisone into the pedal joint. It is presumed that from there the cortisone diffused into the navicular bone (Colles, 1982). Bishop (1960), using this technique in 22 horses with navicular disease, noted that 16 of them "became serviceable".

In 1961 Collins recommended a new approach to the navicular

bursa which was through the deep flexor tendon and onto the flexor surface of the navicular bone, although he could give no results using this technique. Dean (1964) concluded, from intra-bursal cortisone injections in 27 horses with navicular disease, of which 13 were sound and had stayed sound for periods between 4 months and 5 years, that if no response occurred to 2 injections then it was unlikely that there would be any response to this treatment. Intra-bursal injections of corticosteroids has also been reported by Wintzer (1964), Baum (1974) and Adams (1974). Adams (1974) maintained that this form of therapy was of little value as a permanent cure. It was noted by Colles (1982) that the technique of injection into the navicular bursa resulted in trauma to the flexor cartilage of the navicular bone and possibly adhesions between the deep flexor tendon and the navicular bone. It has been suggested that the response to intra-bursal corticosteroid injection was as a result of reduced inflammation and capillary permeability which in turn could relieve congestion and so partial ischaemia in the navicular bone (Colles, 1982).

OTHER FORMS OF THERAPY

Injections of Lugol's iodine into the navicular bursa was suggested by Frost (cited by Donahue, 1935) but this form of treatment was not recommended by Adams (1974) since it was thought to be painful and had little therapeutic effect.

Wintzer (1964) treated 7 horses with anabolic steroids and 2 horses with x-ray therapy, with no success. Adams (1974) reported that x-ray therapy did not give consistent results when used for treating navicular disease.

The use of long acting local anaesthetic to block the palmar digital nerves, the effect lasting 4-5 weeks, was reported by Baum (1974). The results of these nerve blocks were not always successful.

ANTICOAGULANT THERAPY

Following the findings of Colles and Hickman (1977) of thrombosis and arteriosclerosis in the distal navicular arteries of navicular disease cases, and the proposed theory of progressive ischaemia being involved in the pathogenesis of navicular disease, therapy using the oral anticoagulant warfarin was initiated by Colles (1979a). He considered that anticoagulants could be beneficial in the early stages of navicular disease by allowing a secondary periosteal blood supply to develop.

Warfarin was the anticoagulant of choice for this work because it could be administered orally, its effect could be quickly and easily monitored using Quick's one stage prothrombin time (OSPT) (Biggs and Macfarlane, 1962), it has a specific antidote Vitamin K₁, and it was of low cost. Warfarin is widely used in man for long term therapy, post operatively and for myocardial infarction.

Colles (1982) reported that over a 3 year period, 77 of the 100 horses with navicular disease treated with warfarin, became sound. Colles (1982) noted that cases which had shown a slow response to warfarin therapy, or which had not become completely sound, tended to be horses less than 5 years old, horses that had been lame for a long period and horses showing a rapid deterioration in the condition. He suggested since 3 horses had remained sound after cessation of warfarin therapy, that warfarin could possibly effect a cure in some cases. Fricker et al. (1982) considered the positive effect of warfarin

therapy in navicular disease was because navicular disease was an obliterating endarteritis, the treatment of which in man is long term anticoagulant therapy.

COMPARISON OF THE DIFFERENT TREATMENTS

There are few comparative reports of treatments for navicular disease. Wintzer (1964) compared 5 different treatments in 491 cases of navicular disease, and 89 untreated cases of navicular disease as controls. The treatments compared were corrective shoeing, neurectomy, cortisone therapy, anabolic steroid injections and x-ray irradiation. After one year only 60 horses were still usable and of these only 28(4.5%) were considered to be completely sound. He concluded that no particular treatment was any more successful than the others.

Ackerman et al.(1977) compared neurectomy with a combination of analgesics and corrective shoeing but found no significant difference in the success rate between these. He noted that only 9 out of the 38 horses treated were sound 6 months later.

Colles (1982) examined corrective shoeing, neurectomy, cortisone therapy, analgesics and anti-inflammatory agents, for the treatment of navicular disease in a small number of horses. His results confirmed the findings of Wintzer (1964).

From the literature it is evident that the majority of treatments for navicular disease are palliative and based upon the assumption that navicular disease is irreversible in nature. The recent innovation of anticoagulant therapy is aimed at treating the proposed underlying pathology of the condition.

INTRODUCTION

In view of the recent introduction of anticoagulant therapy of navicular disease, it was decided to carry out a comparative study of the different forms of treatment for navicular disease in current use, with particular emphasis on oral warfarin therapy.

Of the 151 cases of navicular disease (Group B) referred to the Department of Surgery, R.(D.)S.V.S. over the 3½ year period of this study, 120 of them have been treated. Each horse received one of six forms of treatment, and some of these horses received more than one form of treatment.

The following treatments were evaluated:

- (i) Rest.
- (ii) Oral warfarin therapy.
- (iii) Corrective shoeing.
- (iv) Phenylbutazone.
- (v) Neurectomy.
- (vi) Isoxsuprine hydrochloride.

As far as possible the treatment to be given to each horse was selected on a random basis, but because of the clinical nature of this study and because all of the horses with navicular disease were referred cases, this could not be strictly adhered to. The selection of the type of treatment was therefore influenced by the type of treatment, if any, the horse had received prior to referral, and the wishes of the owner.

The results of treatment were assessed both clinically and radiographically using the navicular scoring technique, since the navicular score had proved a useful method of assessing the stage of the disease process.

MATERIALS AND METHODS

At the initial examination of each horse, regardless of the type of treatment to be given, the horse was examined clinically and the degree of lameness assessed (APPENDIX 2.2). A routine radiographic examination was then carried out, a minimum of 2 antero-posterior views were taken of each foot, and the navicular score calculated from these as described previously.

In most cases the horse was not admitted to the R.(D.)S.V.S. veterinary hospital, but treated as an outpatient. The owner and the referring veterinary surgeon were given suitable instructions as to the course of treatment, and this was carried out under the supervision of the referring veterinary surgeon.

Each horse was re-examined at 3-4 month intervals depending on the course of treatment, or in the case of the horses given rest, re-examination was carried out at the end of the rest period.

At each re-examination the lameness was assessed clinically, routine radiographic examination carried out, and the navicular score calculated.

The results of the clinical assessment were recorded as:

Sound - this was recorded when there was no evidence of lameness on clinical examination and no response to pressure with hoof testers or to the flexion test. Some of the horses which met these criteria had been returned to the work carried out prior to the onset of lameness.

Definite improvement in lameness - was recorded in cases in which there was an improvement in the lameness from the initial examination. In some cases the improvement in lameness was

sufficient to allow the horse to perform light work.

No change in lameness - was recorded when the degree of lameness had shown no change from that of the initial examination.

Increase in severity of lameness - this was recorded when the horse showed an increase in the degree of lameness from that recorded at the initial examination.

The treatment regimes were as follows:

REST

Twenty-nine horses with navicular disease were given a period of rest of not less than 3 months duration. They were either, turned out to grass, or turned out during the day and stabled at night depending upon the season, in order to allow some measure of exercise.

No change was made to the shoeing of these horses.

The horses were re-examined at the end of the rest period.

ORAL WARFARIN THERAPY

The use of the oral anticoagulant warfarin for the treatment of navicular disease in horses was introduced by Colles (1979a) following his findings of arterial thrombosis and arteriosclerosis in the arteries of the navicular bone in navicular disease cases.

Sodium warfarin is a coumarin derivative whose main pharmacological action is the inhibition of blood clotting mechanisms, by interference with the synthesis of clotting factors, II (prothrombin) VII, IX and X (Levine, 1975). Warfarin produces this action by antagonism of Vitamin K which is required for the synthesis of these factors. As well as its effects on clotting factors, warfarin has been shown to cause vasodilation of the coronary arteries in pigs

in vitro (Blake, Wood, O'Moore and Neel, 1962) and to aid in recanalisation of experimentally induced thrombosis of the femoral artery in rabbits, (Wright, Kubik and Hayden, 1953). The effects of warfarin on the clotting mechanisms in the horse were examined by Archer (1959), the haematological and blood enzyme values following oral administration of warfarin to ponies were studied by Scott, Sandler and Byars (1979) and the pharmacokinetics of warfarin in the adult horse were reported by Thijssen, Van den Bogaard, Wetzel, Maes and Muller (1983).

Colles (1982) used oral warfarin therapy in the treatment of 100 horses with navicular disease. The warfarin was administered once per day in the food, and the one stage prothrombin time (OSPT) of each horse estimated at intervals. The required increase in prothrombin time was between 2 and 4 seconds longer than the established normal value. The dose rate required to produce this effect in horses ranged from 0.012 mg/kg to 0.17 mg/kg bodyweight.

In this present study 60 horses with navicular disease were treated with oral warfarin. In the early stages of the work, horses to receive oral warfarin therapy were admitted to the R.(D.)S.V.S. veterinary hospital, so that the overall effects of treatment could be assessed. The horses were discharged once stabilised on the required dose of warfarin.

All of these horses were housed in loose boxes, and exercised daily. The type and amount of exercise given to each horse was dependent on the degree of lameness, and was increased if the lameness improved.

The protocols used for oral warfarin therapy are those described by Colles (1979a).

Following admission to the veterinary hospital a blood sample was taken from each horse on 2 consecutive days in order to establish the normal OSPT for the individual horse. Blood samples for OSPT estimation were taken into commercially available* 2 ml blood sample bottles with 4 mg of [Wintrobe] mixture⁺ added as the anticoagulant.

Estimation of the one stage prothrombin time was carried out using the techniques described by Biggs and Macfarlane (1962). For this test a commercially available rabbit tissue thromboplastin-calcium reagent[#] was used.

All OSPT estimations were carried out by the same person in order to achieve consistency of results.

The normal OSPT for each horse was calculated by taking the average of the one stage prothrombin times of the 2 blood samples.

Blood samples were taken at intervals for routine haematological examination and determination of amino aspartate transferase (AST) and γ -glutamyl transpeptidase (γ GT) levels.

The dose of warfarin to be administered to each horse was calculated at the initial dose rate of 0.018 mg/kg bodyweight. The calculated dose of warfarin was then administered in tablet form**, at approximately the same time, once per day, in the food.

Blood samples for further OSPT estimations were collected at 5 day intervals following the start of treatment. As recommended

* Teklab (Medical Laboratories Ltd.), Sacriston, Co. Durham, England.

+ Ammonium and Potassium Hydroxide.

Simplastin, General Diagnostics, Warner-Lambert (U.K.) Ltd., Eastleigh, Hants., England.

** Marevan, Duncan Flockhart and Company Ltd., London, England.

by Colles (1979a) if no increase in OSPT was recorded after 10 days treatment, the dose of warfarin was increased by 20% of the initial calculated dose, and blood sampling for OSPT estimation continued at 5 day intervals. When 3 consecutive blood samples showed an increase in OSPT to the desired level, the interval between blood samples was increased to 1 week. If the OSPT remained at the desired level, the horse was considered to be stabilised on warfarin. In most stabilised cases the OSPT estimation was carried out at monthly intervals.

Once a horse was stabilised on the required dose of warfarin it was discharged from hospital, and blood samples were taken at the required intervals and sent to the Department of Surgery for estimation of OSPT.

Colles (1979a and 1982), established that if blood samples were sent by post as whole blood and not as serum, this did not adversely affect the OSPT of horses provided this period did not extend beyond 5 days.

It was found in many of the hospitalised cases that, following return to their home environment, the dose of warfarin had to be altered in order to maintain the required increase in OSPT. In view of this finding, in the later stages of this work horses to receive warfarin therapy were no longer hospitalised during stabilisation, but were treated as out patients. The dose of warfarin required to achieve stabilisation was established with the co-operation of the referring veterinary surgeons.

All of the horses receiving oral warfarin therapy were returned to the Department of Surgery at 3-4 month intervals for re-examination, clinically and radiographically and their navicular scores were calculated.

CORRECTIVE SHOEING

Corrective shoeing was used as the only form of treatment in 10 horses with navicular disease. The type of shoe applied to both fore feet in these cases was similar to the shoes described by McCunn (1951) and Hickman (1964 and 1977). The shoes had rolled toes and raised or wedged heels, the heels being raised no more than $\frac{3}{4}$ of a centimetre. The farrier encouraged the foot to become more upright by paring the foot well back at the toe and allowed the heels to grow.

The feet were pared and re-shod at 6 week intervals.

The horses receiving corrective shoeing were exercised daily in the same manner as horses receiving other treatments, the type and amount depending on the severity of lameness and the response to treatment.

Clinical and radiographic examinations were carried out at 3-4 month intervals, and the navicular scores calculated.

ANALGESICS

Phenylbutazone was administered to 8 horses with navicular disease over various periods of time. The dose administered was that required to allow the individual horse to perform the type of work being carried out prior to the onset of lameness.

It was possible to carry out re-examinations of only 5 of these horses, which were carried out at 3-4 month intervals and the navicular score calculated.

No change was made to the shoeing of these horses.

NEURECTOMY

Low palmar digital neurectomies were performed on 3 horses

with navicular disease, one unilaterally and 2 bilaterally.

The neurectomies were carried out with the horse under general anaesthesia, following the method described by Adams (1974). The procedure of epineural capping as described by Evans (1970) was not included.

Sections at least 12 mm in length were removed from the medial and lateral palmar digital nerves.

Pressure bandages were applied for a week after the operation during which time the horse was confined to a loose box. After this the horse was walked out in hand for several days, and the amount of exercise was slowly increased.

Clinical and radiographic re-examination was carried out in 2 out of the 3 horses treated in this manner.

ISOXSUPRINE HYDROCHLORIDE

Isoxsuprine hydrochloride is a beta adrenergic agonist whose principal effect is to increase muscle blood flow by acting directly and selectively on the smooth muscle of blood vessel walls (Samuels and Shaftel, 1959; Lowe, 1983).

It has been used in man for the treatment of intermittent claudication associated with peripheral vascular insufficiency, and for the treatment of night cramps and Raynaud's disease, with variable degrees of success (Samuels and Shaftel, 1959; Strandness, 1970).

The first report of the use of isoxsuprine hydrochloride in the treatment of navicular disease in the horse was by Rose, Allen Hodgson and Kohnke (1983). They reported an 83% success rate

following treatment with isoxsuprine hydrochloride, of 27 horses with navicular disease.

In this present study a small trial was carried out using isoxsuprine hydrochloride* to treat 10 horses with navicular disease according to the protocols described by Rose et al. (1983).

The first 4 horses receiving this form of treatment were hospitalised for the full course of treatment so that the overall effects of the vasodilator could be assessed. The isoxsuprine hydrochloride was administered in tablet form at a dose rate of 0.6 mg/kg bodyweight, given twice daily in the food for 10 weeks, followed by once daily for 2 weeks.

Blood samples were taken prior to the start of treatment, and at 2 week intervals throughout the course of treatment, for routine haematological examination, and determination of AST and γ GT levels.

The remaining 6 horses were discharged after the initial clinical and radiographic examination, and treatment carried out as described previously, under the supervision of the referring veterinary surgeons.

All of the horses receiving isoxsuprine hydrochloride were exercised daily as described previously, the amount of exercise being related to the degree of lameness.

The horses were re-examined clinically and radiographically, and their navicular scores calculated at the end of the 12 week course of treatment.

* Duvadilan, Duphar Ltd., Southampton, England.

The differences between the groups of horses receiving different treatments, in the duration of lameness, the degree of lameness, and their navicular scores, prior to the start of treatment, were assessed by analysis of variance. The 3 horses receiving neurectomies were not included since the numbers were considered too small for statistical analysis.

The results of the different types of treatment were compared and statistically analysed for significance against chi-squared distribution. The results of treatment with phenylbutazone and neurectomy were not included in this statistical analysis, since the numbers were considered insufficient.

RESULTS

The results of the different forms of treatment of navicular disease used in this study are contained in APPENDICES 5.1 - 5.5.

No significant difference was found between the 5 groups of horses, in the duration of lameness, the degree of lameness prior to the start of treatment, and the navicular scores prior to the start of treatment (TABLE 5.1). The groups were therefore considered suitable for a comparative study of treatments.

REST

The results of this treatment of 29 horses with navicular disease are shown in TABLE 5.2. In this table the horses are grouped into 1 of 5 groups, according to their navicular score prior to the start of treatment. The results of treatment were recorded at the end of the rest period, the rest periods varying from 3 months to 6 months, and in no case was the period of rest less than 3 months.

A return to soundness, or a definite improvement in the lameness as defined earlier, were considered as a positive response to the treatment.

A positive response to rest was recorded in 10(34.5%) of the horses in this group, 6(20.7%) of them showing a return to soundness.

These 6 horses were returned to the work being carried out prior to the onset of lameness, 3 of them have remained sound for more than 1 year following the rest period.

In the group of horses rested, the navicular scores prior to the start of treatment ranged from 13.5 to 34, and horses from each of the 5 navicular score groups were treated (TABLE 5.2).

A greater percentage of the horses treated in the 10-14 navicular score group showed a positive response to rest, than in the other navicular score groups. (FIG 5.1).

At the end of the rest period the navicular score was again calculated and an increase in navicular score was found in 16 of the 29 horses. No change was found in the navicular score of the other 13 horses. The navicular score did not decrease in any of the cases examined.

ORAL WARFARIN THERAPY

The results of oral warfarin therapy in 60 horses with navicular disease are given in TABLE 5.3, in which the horses are grouped into 1 of 5 groups according to their navicular score prior to the start of treatment.

The results of warfarin treatment in this study were all recorded from horses that had been receiving oral warfarin for periods of 4 months or longer except for 3 horses from the fluorochrome bone labelling experiment*. Two of these horses received warfarin for 2 months and 1 horse received warfarin for only 1 month (TABLE 4.5).

Since warfarin therapy is a long term treatment, and the response to treatment can change over a period of time, the results recorded in TABLE 5.3, are the results from the last examination of each case.

The dose rate of warfarin required to produce an increase in OSPT of 2-4 seconds ranged from 0.018 mg/kg to 0.08 mg/kg bodyweight.

* Chapter 4, Part II.

A positive response to oral warfarin therapy was recorded in 32 (53.3%) of the horses in this group. Of the horses showing a positive response 23 (38.3%) were considered to be sound. These horses had been sound for different lengths of time extending to 2 years, following the start of warfarin therapy.

The navicular score in this group of horses prior to the start of treatment ranged from 11 to 35, and horses from each of the 5 navicular score groups were treated. A greater percentage of the horses from the lower navicular score groups became sound, or had shown a positive response to the treatment than from the higher navicular score groups (FIG 5.1).

Of the horses treated in the higher navicular score groups, a greater percentage showed an increase in severity of lameness than in the lower navicular score groups.

The horses receiving warfarin were re-examined at 3-4 month intervals.

Five of the horses showed an initial positive response to treatment, either sound or a definite improvement in lameness, at the first re-examination, but at subsequent re-examinations it was found that the lameness had returned, and in 3 of these horses the severity of lameness had increased.

Of the 23 horses considered sound following treatment, 17 were noted sound at the first re-examination 4 months after treatment commenced. Five of the remaining 6 horses became sound from 4 to 8 months after the start of treatment, and 1 became sound 8-12 months after the start of treatment.

Two of the treated horses showed a positive response before the OSPT had been increased to the desired level.

The administration of warfarin was ceased, after 12 months of treatment, in 2 of the horses that had become sound (B31 and B39). Both of these horses have remained sound after cessation of warfarin therapy.

At each re-examination the navicular score was calculated from the routine radiographs.

In 8 of the horses that had become sound while receiving warfarin therapy, no change was found between the pre-treatment navicular score and the navicular score at the last examination, while in the other 15 horses that were sound the navicular score had increased by 0.5 to 4.5, from that recorded pre-treatment. (FIG 5.2).

No change in navicular score between the pre-treatment examination and the last examination was recorded in 1 horse showing definite improvement in lameness. The other horses showing definite improvement in lameness had increases in their navicular scores ranging from 0.5 to 2.

Of the 20 horses in which no change in lameness had been noted, 5 had no change in their pre-treatment navicular score at the last examination, and the remaining 15 had increases from 0.5 to 6 in their navicular score from the pre-treatment level.

The horses showing an increase in the severity of lameness all had an increase in their navicular score from that recorded pre-treatment. The increase in their navicular score ranged from 1.5 to 17. (FIG 5.3).

The navicular score did not decrease in any of the cases examined in this study.

Oral warfarin therapy is a long term treatment, and since all of the horses receiving warfarin therapy in this study had been

treated for different lengths of time when these results were recorded, the navicular score changes noted have little meaning unless they are examined in relation to the length of time between pre-treatment scoring and those recorded at the last examination.

Of the 8 sound horses in which there was no change in navicular score from that recorded prior to treatment, 3 had been treated for 4 months, 3 for 8 months, 1 for 12 months and 1 for 16 months. One horse with definite improvement in lameness had no change in its navicular score and this was recorded after 4 months of treatment. Five horses in which no change in lameness had been recorded, also had no change in their navicular scores after 1-8 months treatment.

In order to compare the increases in navicular scores between the horses that had become sound, those showing improvement in lameness, those with no change in lameness, and those in which there was an increase in severity of lameness, the increase in the navicular score was divided by the number of months between the start of treatment and the last examination, in each case. This calculation gave a notional increase in navicular score per month for each horse. From these figures the mean and standard deviation of the increase in navicular score per month for the groups of horses were calculated, and the differences between the groups assessed by analysis of variance (TABLE 5.4).

No difference was found in the increase in navicular score per month between the horses becoming sound and those showing a definite improvement in lameness, but a highly significant difference was found between those horses showing a positive response to treatment and those showing no change in lameness or an increased severity of

lameness. A greater increase in navicular score per month was recorded in those horses showing no change or increased severity of lameness than in those showing a positive response to treatment.

A significantly greater increase in navicular score per month was found in the group of horses showing an increased severity of lameness than in those horses showing no change in lameness.

No change in the routine haematology, AST and γ GT levels was recorded either during or after the stabilisation phase of oral warfarin therapy in any of the horses examined.

In 2 of the horses receiving oral warfarin therapy adverse reactions occurred. One apparently stabilised horse died as a result of severe intra-abdominal haemorrhage following an attack of colic, and the other horse excreted blood in its faeces for several days during the stabilisation phase, which resolved following cessation of warfarin treatment. The OSPT recorded of the latter horse when it first started excreting blood was 2.5 times its normal OSPT. Warfarin treatment was withheld for 10 days and then administered to the horse as before but at a lower dose.

CORRECTIVE SHOEING

The results of this form of treatment of 10 horses with navicular disease are contained in APPENDIX 5.3. In table 5.5 the horses are grouped into one of 5 groups according to their navicular score prior to the start of treatment.

Re-examination of the horses treated in this manner was carried out at 3 - 4 month intervals.

A positive response to corrective shoeing was recorded in 5(50%) of the horses, 2(20%) being considered as sound. An increase

in the severity of lameness was not recorded in any of the horses in this group.

A greater percentage treated from the 10-14 navicular score group have shown a positive response to treatment, than from the other navicular score groups. (FIG 5.1).

When the navicular scores pre-treatment and the navicular scores after 3-4 months treatment were compared, no change was found in the horses considered to be sound, in 1 out of 3 horses showing an improvement in lameness and in 3 horses in which no change in lameness was recorded. Although the numbers were too small for statistical analysis, very little difference was found, in the increase in navicular score between the remaining 4 horses.

The 2 horses that were sound at the re-examination have both remained sound for periods of 9 months and 1 year, and are still being correctively shod.

PHENYLBUTAZONE

Although 8 of the horses with navicular disease were administered phenylbutazone, re-examinations have only been possible in 5 of them. The results of these re-examinations are contained in APPENDIX 5.4.

This form of treatment differed from most of the other forms of treatment in this study, since all 8 horses showed a positive response to treatment as long as the treatment was maintained. In all cases the horses became lame when the phenylbutazone was withdrawn.

Two of the 5 horses re-examined ceased to show a positive response to phenylbutazone, despite increasing doses, after 6 months

of treatment in one case, and 14 months treatment in the other.

All 5 cases re-examined showed an increase in their navicular score as compared to the pre-treatment navicular scores. The calculated increase in navicular score per month in this group of horses was similar to that found in the horses receiving warfarin therapy showing an increase in severity of lameness. Statistically using the students' 't' test no significant difference was found between the 2 groups of horses in the increase in navicular score per month ($t = 0.07$; d.f. = 11; $P > 0.99$).

NEURECTOMY

Following low palmar digital neurectomy in 3 horses, one unilaterally (B97) and 2 bilaterally (B52, B135), all 3 horses appeared to be sound 1 week later. Within 1 month of neurectomy one horse (B135) became lame on the left fore foot, and the lameness gradually increased. The return of lameness was considered to be due to the formation of painful neuromas on the end of the sectioned nerves. The horse was subsequently destroyed and the results of post mortem examination are given in APPENDIX 4.2.

The other horse, in which bilateral neurectomy was performed, remained sound and was able to carry out the work done prior to the onset of lameness, for 14 months following neurectomy, at which time the lameness returned.

Radiographic examination of both fore feet in this case revealed an increase in the navicular score from 22 prior to neurectomy to 29.

ISOXSUPRINE HYDROCHLORIDE

The results of treatment with isoxsuprine hydrochloride in 10 horses with navicular disease are given in TABLE 5.6, in which the horses are grouped into 1 of 5 groups according to their navicular score prior to the start of treatment.

The results of this treatment were all recorded at the end of the course of isoxsuprine.

Following treatment with isoxsuprine hydrochloride 3(30%) horses were considered to be sound, 2(20%) showed a definite improvement in lameness, 4(40%) showed no change in lameness, and in 1(10%) horse the severity of lameness had increased. Fifty percent of the horses treated with this vasodilator had therefore shown a positive response.

The 3 horses sound at the end of the course of treatment, remain sound, and continue to work 6 months post treatment.

When the navicular scores were calculated at the end of the treatment course, no change in navicular score from that calculated prior to the start of treatment was found in 1 horse that had become sound, 1 horse showing a definite improvement in lameness, and in 2 horses in which there was no change in lameness. The other 5 horses all showed an increase in their navicular scores.

Although the figures are too small for statistical analysis, the greatest increase in navicular score over the period of treatment was found in the horses showing an increased severity of lameness.

Blood samples were taken at 2 week intervals throughout the course of treatment in 4 horses, and no change was found in routine haematology, AST and γ GT levels.

Isoxsuprine appeared to be well tolerated in all cases, and no

adverse reactions were noted.

COMPARISONS OF THE DIFFERENT FORMS OF TREATMENT

No significant difference was found in the results of treatment with rest, oral warfarin therapy, corrective shoeing and isoxsuprine hydrochloride, in all 109 horses with navicular disease. (TABLE 5.7) The results of oral warfarin therapy included in this analysis were the results recorded at the last examination in each case, and, this varied from 1 to 24 months after the start of treatment, whereas the results of rest, corrective shoeing and isoxsuprine hydrochloride included in the analysis, were those recorded at re-examinations 3-4 months after the start of treatment.

Further statistical analysis was carried out to assess both the short term and long term results of the different treatments. The number of horses recorded as sound at the first re-examination 4 months after the start of treatment with warfarin therapy, and the number of horses recorded as sound 3-4 months after the start of the other forms of treatment, were compared and the results statistically analysed for significance using chi-squared distribution.

Similar comparisons were made at 6-8 months and at 1 year after the start of treatments (TABLE 5.8).

Treatment with isoxsuprine hydrochloride was not included in the analysis of the results of treatment after 1 year, because the long term results are not yet available.

No significant difference was found between the results of the different forms of treatment at 3-4 months and at 6-8 months after the start of treatment. However, 1 year after the start of treatment, significantly more horses receiving oral warfarin therapy were sound than horses that had received other forms of treatment.

DISCUSSION

From the review of the literature it is evident that there are a number of different forms of treatment for navicular disease in the horse. When such a number of different treatments exist for a disease, this may be a reflection of a poor overall success rate.

Comparative studies of some of these treatments have been carried out (Wintzer, 1964; Ackerman et al., 1977; Colles, 1982).

In order to compare objectively the results of different treatments of a disease either:

- (i) one group of affected animals is used, and each animal in the group is given each of the different treatments, and therefore comparisons are made within subjects.

or,

- (ii) different groups of affected animals are used, and each group is given a different treatment. Treatment comparisons are therefore made between subjects (Armitage, 1960).

Using the first method of comparing treatments eliminates variations in response from one animal to another, but after cessation of one form of treatment the animal would have to return, or be returned to its pre-treatment disease status, before another form of treatment could be administered. This therefore requires that the disease being treated is non-progressive or only slowly progressive, and that either the different forms of treatment are palliative with a suitable time period between each one, or that the disease processes can be experimentally produced.

Using the second method of comparing treatments, in order that the comparison between treatments should be valid, the different

groups of animals would require to be at the same stage of the disease process, but despite this individual variations in response may affect results.

Navicular disease has, as yet, not been produced experimentally and therefore all cases of navicular disease to be treated are naturally occurring cases. Navicular disease is a chronic progressive condition, and as has been shown from radiographic and pathological examinations earlier in this study, any group of horses with navicular disease will present a varied range of progressive pathological changes.

For these reasons, and because of the clinical nature of this work, the first method of comparing treatments was not suitable, and the second method described was used in this study. Groups of horses with navicular disease were given different forms of treatment.

As horses with navicular disease at different stages in the disease process would be present in each group, it was important to establish whether the groups were suitable for comparison. The factors considered for this purpose were the mean duration of lameness, degree of lameness, and navicular score for each group of horses prior to the start of treatment, since these were considered to be the best available measures of the disease status of the horses.

No significant difference was found between the groups of horses in these factors and therefore the groups were considered suitable for a comparison of treatments.

Numerous workers have assessed the results of different treatments of navicular disease using clinical criteria (Wintzer, 1964; Ackerman et al., 1977; Colles, 1979a, 1982; Rose et al., 1983).

In this present study the results of the different treatments were assessed both clinically and radiographically.

The clinical assessment of the results of treatment was carried out at intervals throughout the treatment and/or at the end of treatment. The results of treatment were defined as, sound, definite improvement in lameness, no change in lameness, or increased severity of lameness, each of these assessments being related to the degree of lameness recorded at the initial pre-treatment examination. As in the assessment of the initial degree of lameness shown by an animal, the assessment of changes in the degree of lameness is also a highly subjective process. In order to maintain consistency, as in chapter 2 of this work the assessment of the degree of lameness at the initial clinical examination, and the assessment of the results of treatment at the subsequent clinical examinations, was carried out by one person.

The radiographic assessment of the results of treatment was based on the navicular scoring technique described earlier. Since it has been shown that the calculated navicular score was correlated to the degree of pathological change in the navicular bone, and that the navicular score could change in navicular disease cases, the navicular scoring technique was considered a suitable method of:

- (i) establishing the stage of the disease process to be treated in each case.
- (ii) assessing the results of treatment related to the clinical findings.
- (iii) comparing the results of treatment, when administered at different stages in the disease process.

In chapter 3 of this study all the horses examined radiographically were classified into 1 of 7 groups according to their navicular score and it was found that in the navicular disease cases the navicular scores were distributed within 5 groups from 10-14 to 30+.

These 5 navicular score groups have been used to identify the different stages of the disease process treated within the different treatment groups.

REST

Rest as a form of treatment of navicular disease has been suggested by several workers (Reeks, 1925; Hickman, 1964; Johnson, 1982). In contrast Turner (1829), Smith (1886) and Pryer (1934) maintained that rest predisposed horses to navicular disease, particularly if they were confined in a stable.

All the cases of navicular disease given a period of rest in this study were either turned out to grass, or turned out during the day and stabled at night depending upon the season. This method of management ensured the horses were exercised daily.

At the end of the rest period 6 out of the 29 horses rested had shown a return to soundness. These 6 horses were subsequently returned to work, after 1 year 3 of the 6 remained sound. Although a 20% return to soundness was recorded at the end of the treatment, only 10% of the original 29 horses treated were sound 1 year later.

In this study a greater percentage of horses in the lower navicular score groups showed a positive response to rest. From these results it is considered that a horse with navicular disease will show a better response, to treatment with a period of rest,

if treated in the early stages of the disease than if treated in the later stages of the disease.

These results support Hickman's (1964) suggestion that 3-4 months rest at grass in the early stages of navicular disease could relieve the condition for a period of time in some cases.

Radiographic assessment of treatment showed that at the end of the rest period an increase in navicular score was found in 55% of the horses, and no change in navicular score was detected in the other 45%. No relationship could be found between the clinical response to treatment with rest and the changes in navicular score recorded.

The increase in navicular score per month was calculated for this group of horses, as described in the section on results of treatment with oral warfarin therapy, and when analysed statistically, no significant difference was found in the increase in navicular score per month between the horses showing a return to soundness, those showing a definite improvement in lameness, and those showing no change in lameness. (TABLE 5.9)

It is suggested from these radiographic findings that either rest as a treatment for navicular disease has little effect on the disease process, or, the number of horses showing a positive response to treatment is too small to show significant changes and/or the period over which the increase in navicular score per month was calculated is too short to demonstrate any significant differences.

ORAL WARFARIN THERAPY

Oral warfarin therapy is considered to be a long term form of treatment for navicular disease, based on the hypothesis that

navicular disease is the result of obstructive vascular changes within the navicular bone which give rise to areas of partial ischaemia (Colles, 1982). The aim of the treatment is to allow a secondary periosteal blood supply to develop without thrombosis occurring.

Colles (1979a) considered that there was a relationship between the age of the horse, the duration of lameness, and the time to recovery, which he expressed in the form of an equation.

Applying this equation to a number of horses to be treated with oral warfarin therapy in this present study, it was found that the longest time to recovery should be 16 weeks. For this reason it was decided to allow this period of time before recording the results of the first re-examination to allow comparison with other treatments in which there was a similar time period before the first re-examination.

A positive response to oral warfarin therapy was recorded at the last examination in 53.3% of the cases treated, and a return to soundness was recorded in 38.3% of the cases treated. Of the horses recorded as sound, 74% were sound at the first re-examination at 4 months. It would therefore appear that the majority of horses which will respond to oral warfarin therapy, do so in the first 4 months of treatment. However, failure to show a positive response in the first 4 months of treatment does not mean that a horse will not respond at a later date, since in this study 5 horses became sound between 4 and 8 months of treatment, and 1 horse became sound after 8 - 12 months treatment.

A number of cases showed an initial positive response to treatment at the first re-examination, but the lameness had returned

by the following re-examination.

One possible explanation for this finding is that the initial positive response in these cases may not be due to the effects of warfarin therapy, but may be related to the management of the horses during this period. In the first few months of warfarin therapy, especially during the stabilisation phase, these horses will be given "light exercise" which is similar to the management of the rested horses.

In this study a better response to treatment with oral warfarin therapy was recorded from the horses in the lower navicular score groups than from those in the higher navicular score groups. No horse in the 30+ navicular score group, showed a positive response to treatment with oral warfarin therapy, and 3 of the 4 horses in this navicular score group showed an increased severity of lameness over the period of examination.

Colles (1982) suggested that better results would be achieved by using oral warfarin therapy at an early stage of navicular disease, and that if severe degenerative changes had occurred in the fibrocartilage of the navicular bone, then treatment was unlikely to be successful.

The results of this present study, using navicular scores to establish the stage of the navicular disease process before treatment, supports these findings of Colles (1982).

Since, as was stated earlier, all of the horses receiving warfarin therapy had been treated for different lengths of time when the last navicular scores were recorded, absolute changes in navicular score are meaningless, unless related to the length of treatment. It is therefore considered that the increase in navicular

score per month is a more appropriate method of assessing radio-graphically the results of treatment.

From statistical analysis it was found that in horses in which an increase in navicular score was recorded, a return to soundness was accompanied by a significantly lower increase in navicular score per month, than in those horses showing no change in lameness or increased severity of lameness. In all horses showing an increased severity of lameness there was a significantly greater increase in navicular score per month than in those horses in which there was no change in lameness.

No change in navicular score was recorded in some horses showing a positive response to treatment. However, this finding was not solely a feature of horses responding positively to treatment, and in some horses, in which there was no change in lameness, also had no change in their navicular score.

The possible reasons for the navicular score remaining constant in these horses are:

- (i) that the condition had ceased to progress, or was in a non-progressive phase.
- (ii) that the period over which the navicular scores were recorded was not long enough to detect changes.

The period of treatment over which the navicular score had remained unchanged was calculated for each of these horses, and it was found that the navicular score was unaltered over a significantly longer period in the horses showing a return to soundness than in the horses in which there was no change in lameness. ($t = 2.35$; d.f. = 11; $P < 0.05$)

It is therefore concluded from these radiographic findings that in the horses with navicular disease, showing a return to soundness while receiving oral warfarin therapy, the condition may become non-progressive or there may be a slower rate of progression of the disease processes than in the horses not showing a positive response to treatment.

The overall results of treatment of navicular disease with oral warfarin therapy in this study fail to confirm the findings of Colles (1982), who reported that 77 horses out of 100 treated with warfarin had shown a return to soundness.

This difference in the results of treatment with oral warfarin therapy may be due to:

- (i) differences in the stage of the disease processes being treated, the greater the number of early navicular disease cases treated, the better the chance of positive results.
- (ii) concurrent corrective shoeing. Colles (1982a) reported that corrective shoeing and regular exercise had been included with oral warfarin therapy in the treatment regime.

Several features noted by Colles (1982) in his warfarin therapy trial have also been recorded in this present study.

These are:

- (i) a small number of horses showed a positive response to treatment before the OSPT was increased to the desired level.
- (ii) a number of horses have shown an initial positive response to treatment, but lameness has subsequently returned.
- (iii) in a number of horses showing a positive response to treatment, withdrawal of warfarin resulted in a return to lameness within 24-48 hours.

- (iv) in other cases showing a positive response to treatment, withdrawal of warfarin had little effect on the response. Two horses had warfarin therapy withdrawn after 1 year's treatment and have remained sound.

The rationale behind using anticoagulants in the treatment of navicular disease in the horse was based on evidence of occlusive vascular disease in the navicular bones of navicular disease cases and the subsequent hypothesis of progressive ischaemia involved in the development of navicular disease (Colles and Hickman, 1977; Colles, 1979a). The aim of long term anticoagulant therapy was to allow an adequate secondary periosteal blood supply to develop and to prevent further thrombosis.

In the pathological examinations reported in chapter 4 of this present study, no evidence of occlusive vascular disease was found in any of the navicular bones examined.

It is possible that the positive response to oral warfarin therapy recorded in this study is not due to the anticoagulant effects of warfarin but may be the result of other pharmacological effects.

This may explain why some cases have shown a response to treatment before the OSPT had increased, and the rapid return of lameness following withdrawal of treatment in others.

Other pharmacological effects of warfarin reported include, increased vascular permeability (Kahn, Johnson and De Graff 1971) vasodilation (Blake et al., 1962) and reduction in blood viscosity (Mackie and Douglas, 1978; Colles, 1983).

CORRECTIVE SHOEING

The aims of corrective shoeing in navicular disease cases are:

- (i) in the first instance, to relieve pressure in the region of the navicular bone by displacing the weight from the posterior part of the foot, forward.
- (ii) to correct any underlying abnormalities in foot conformation which may have predisposed to navicular disease, such as the long toe - low heel conformation described in chapter 2.

The first aim of treatment can be achieved by applying corrective shoes, of which there are several different types suggested (McCunn, 1951; Adams, 1974; Østblum et al., 1982 and others). As Colles (1982a) pointed out, in cases in which there is an underlying conformational abnormality, the application of corrective shoes, although they may straighten the hoof-pastern axis, will not correct the conformational abnormality of the foot. To achieve the second aim of this treatment corrective trimming of the hoof should also be carried out.

Ten horses with navicular disease at different stages in the disease process were treated by corrective shoeing. Of these 10 horses 5 showed a positive response to treatment. In all 5 cases an improvement in lameness was noted shortly after the shoes were applied, and 2 of these cases were subsequently found to be sound at the re-examination 3-4 months after the start of treatment. McCunn (1951) emphasised the immediate improvement in lameness that could be achieved by corrective shoeing.

Four of the 5 cases, showing a positive response to treatment had long toe - low heel conformation, which resulted in a broken

hoof-pastern axis. It has been shown that a horse with this type of conformation has a considerably slower rate of blood flow through the foot than a horse with normal foot conformation (Colles, Garner and Coffman, 1979).

The rapid response noted to this form of treatment may be because the slight elevation of the heels, and straightening of the hoof-pastern axis afforded by the corrective shoes, offers immediate reduction in pressure in the region of the navicular bone and may also improve the rate of blood flow through the foot.

In this study 80% of the horses showing a positive response to treatment by corrective shoeing were from the 10 - 14 navicular score group. As with other treatments discussed, there is likely to be a better response to the treatment of navicular disease by corrective shoeing if the treatment is applied early in the course of the disease process.

It is considered, in accord with the findings of Moyer (1979), that in navicular disease, a better response to treatment by corrective shoeing will be recorded in those horses with the poorest foot conformation, since the correction of foot balance will to some extent per se, afford relief.

PHENYLBUTAZONE

The cases of navicular disease treated with phenylbutazone in this study showed a positive response when treatment was administered, and a return to lameness when the treatment was withdrawn. Two of these cases, despite increasing doses of phenylbutazone, returned to lameness after 6 months treatment in one case, and 14 months in the other.

The navicular score, in all cases re-examined, had increased from that recorded at the start of treatment. The increase in navicular score per month was greater in these horses than in horses receiving the other forms of treatment in this study, apart from the horses receiving oral warfarin therapy, in which there was increased severity of lameness.

In accord with Baum (1974), Johnson (1982) and Colles (1982), it is concluded from the results of this study that treatment with phenylbutazone is palliative in nature, since no evidence of suppression of the disease process has been found. Although only a small number of cases have been treated with phenylbutazone, from the results of radiographic examination it is suggested that treatment with phenylbutazone may accelerate the disease processes in navicular disease cases. It is also suggested that one explanation of this result may be the rapid return to normal foot action, due to the analgesic effect of phenylbutazone, causing normal stresses to be applied to a diseased navicular bone.

NEURECTOMY

Since low palmar digital neurectomy was carried out on only 3 horses in this study, the results of treatment are inconclusive.

A review of the literature shows that many authors consider neurectomy to be the only method of "prolonging the useful life" of a horse with navicular disease.

Wintzer (1964), and Ackerman et al. (1977) however, could find no significant difference between neurectomy and other forms of treatment for navicular disease.

In this present study 1 of the 3 horses on which a neurectomy

was carried out has remained sound, and is now being used as a brood mare. One horse developed painful neuromas on the end of the sectioned nerves and became lame again 1 month after neurectomy. The remaining horse showed a return to lameness 14 months after neurectomy. Schebitz (1964) suggested that a return to lameness months or years after neurectomy was either due to the restoration of innervation of the navicular apparatus or as a result of arthritic changes in the digital joints.

Since there are numerous complications associated with palmar digital neurectomy, and it is an irreversible course of action, it is considered, in agreement with Hickman (1964) that neurectomy should only be carried out when the other forms of treatment fail to relieve the condition.

ISOXSUPRINE HYDROCHLORIDE

Following a 12 week course of treatment with isoxsuprine hydrochloride, as described by Rose et al. (1983), in 10 horses with navicular disease, 3 were considered to be sound and 2 showed a definite improvement in lameness. The three sound horses are still sound 6 months after the start of treatment.

All horses showing a positive response to therapy had pre-treatment navicular scores less than 24, and no response to treatment was recorded in the 2 horses with navicular scores greater than 25.

Although only a small number of horses received this form of treatment, and the results of radiographic assessment could not be analysed statistically, no relationship could be established between changes in navicular score and the clinical response to treatment in these horses.

The results of treatment of navicular disease with isoxsuprine hydrochloride in this study fail to confirm the findings of Rose et al. (1983). They reported that 83% of horses treated with isoxsuprine became sound, and 16 out of 19 horses that had treatment discontinued had remained sound for periods between 2 weeks and 10 months. Rose et al. (1983) considered that these results of treatment of navicular disease using a peripheral vasodilator drug supported the proposed ischaemic nature of navicular disease (Colles and Hickman, 1977; Colles, 1982).

Although isoxsuprine is used in man to treat various peripheral vascular disorders, considerable variation in opinion exists as to its efficacy. Samuels and Shaftel (1959) reported increased claudication distance, and increased blood flow, using isoxsuprine hydrochloride in patients with peripheral vascular insufficiency, while in contrast Strandness (1970) found isoxsuprine to be of little use in the treatment of intermittent claudication in man.

Lowe (1983) in a review of vasodilator drugs stated that "there is little sound rationale or proof of efficacy" for using vasodilators in the treatment of peripheral vascular disorders.

The therapeutic aim of vasodilator treatment in disorders in which there is local ischaemia is to selectively increase blood flow in the ischaemic area. Lowe (1983) considered that vasodilators did not produce this effect, particularly since the occlusive nature of the vascular disorders being treated were atherosclerotic rather than as a result of vasospasm.

Since vasodilators will produce generalised vasodilation, they will also in theory reduce blood flow in areas of local ischaemia by diversion of blood through opened vascular channels in other areas

such as muscle (Samuels and Shaftel, 1959; Lowe, 1983).

The effects of isoxsuprine hydrochloride in the treatment of navicular disease may not be as a result of vasodilation, but may be the result of other pharmacological actions.

As does warfarin, isoxsuprine hydrochloride has been shown to decrease whole blood, plasma, and serum viscosity in man (Di Perri, Forconi, Agnusdei, Guerrini, and Laghi Pasini, 1978).

No evidence of occlusive vascular disease or ischaemia was found from pathological examination of any of the navicular bones in this study, but evidence of hyperaemia was found in the navicular bones examined from navicular disease cases. Although the cause of the hyperaemia has not been established, it has been suggested that it is the result of venous stasis in the foot.

It is therefore possible that the effects of warfarin and isoxsuprine hydrochloride in the treatment of navicular disease is the result of their ability to reduce blood viscosity and thus improve blood flow.

COMPARISON OF THE DIFFERENT TREATMENTS

Comparing the results of the different forms of treatment, recorded at the last examination of each horse receiving oral warfarin therapy, and at the examination 3 - 4 months after the start of the other treatments, no significant difference was found. In this comparison no account was taken of the length of time between the start of treatment and the last examination, which in the cases treated with warfarin varied from 1 to 24 months.

In order to compare the short-term results of the different treatments and the long term results of the different treatments, the

number of horses recorded as sound at 3-4 months, 6-8 months, and 1 year from the start of treatment, out of the total number of horses treated, was calculated. Included in this total figure are those horses in which warfarin therapy was withdrawn before 6-8 months or 1 year from the start of treatment because of failure to respond. Three horses in the fluorochrome bone labelling experiment that had received warfarin for periods less than 4 months were not included in these comparisons.

Statistical analysis showed that there was no significant difference between the short term results of treatments at 3-4 months and 6-8 months from their commencement, but significantly more horses receiving oral warfarin therapy than horses that had received other treatments, were sound 1 year from the start of treatment. The long term results of warfarin therapy in this study are better than the long term results of the other forms of treatment.

It is considered that this finding may not be due entirely to the effects of warfarin therapy, but may also be due in part to attention to other factors such as regular exercise and correct although not corrective shoeing.

In addition, the horses showing a return to soundness while receiving warfarin therapy had a significantly lower increase in navicular score per month than the horses showing no response to warfarin therapy.

The increase in navicular score per month in the horses showing a return to soundness after treatment with rest was not significantly different from the horses showing no response to rest, but was significantly greater than the horses showing a return to soundness receiving warfarin therapy ($t = 2.50$; d.f. = 15; $P < 0.05$).

However, the number of horses treated with rest showing a return to soundness and an increase in navicular score was insufficient to draw any conclusions from these results.

Since navicular scores were shown in chapter 4 of this study to be positively correlated to the degree of gross pathological change present in the navicular bone, an increase in the navicular score is likely to be associated with progression of the disease processes. Therefore the results of this study have shown that a return to soundness while receiving warfarin therapy is accompanied by a slower rate of progress of the disease processes than in horses failing to respond to warfarin therapy, or by a cessation in progression of the disease processes.

It is therefore possible that;

- (i) The action of warfarin, or the combined action of warfarin and other factors slows the rate of progress of the disease processes in some horses.

or that;

- (ii) Horses in which there is a slower rate of progress of the navicular disease processes show a better response to treatment with warfarin.

It is also possible that (i) and (ii) may apply to other forms of treatment, and that a return to soundness, regardless of the type of treatment, is associated with a slower rate of progress of the disease processes or by a cessation in the progression of the disease processes.

However, this did not apply to the small number of horses receiving phenylbutazone, in which, in all cases, a high rate of

increase in navicular score was recorded over the period of treatment.

In all the forms of treatment examined in this study, with the exception of phenylbutazone and neurectomy, a better response to treatment has been shown by horses in the lower navicular score groups, in particular in the 10-14, 15-19 navicular score groups. Horses with pre-treatment navicular scores greater than 25 have shown a poor response to any of the forms of treatment administered. It is likely that horses with navicular scores greater than 25 will have severe pathological changes affecting the fibrocartilage surface of the navicular bone and the deep flexor tendon.

These findings support the suggestions of Reeks (1925), Colles (1982) and Østblum et al. (1982), that if treatment were instigated early in the course of the disease, better results would be obtained than if instigated once severe disruption of the fibrocartilage surface of the navicular bone had occurred.

The results of treatment with phenylbutazone or neurectomy do not appear to be related to the pre-treatment navicular scores.

CONCLUSIONS

It is concluded from the results of the different treatments of navicular disease that:

- (i) Navicular scoring before and at intervals during treatment has proved a useful method, when used in conjunction with clinical examinations, of assessing the results of treatment.
- (ii) The short term results of treatment with rest, oral warfarin therapy, corrective shoeing, and isoxsuprine hydrochloride, were similar.
- (iii) The long term results using oral warfarin therapy were significantly better than the long term results with other forms of treatment.
- (iv) A return to soundness in horses receiving oral warfarin therapy was accompanied by a slower rate, or cessation of the progressive disease processes.
- (v) The positive response to treatment with warfarin or isoxsuprine hydrochloride shown by navicular disease cases is less likely to be related to the prevention of thrombosis or vasodilation than to their ability to reduce blood viscosity.
- (vi) Corrective shoeing is an important part of the treatment of navicular disease cases. Although the results of corrective shoeing on its own were not significantly better than the other treatments, since this treatment is aimed at correcting any underlying abnormality in foot conformation, and since there was a rapid response shown in a number of cases, it is considered that corrective shoeing should be included in the treatment of navicular disease cases.

- (vii) Regardless of the type of treatment to be administered, the earlier in the course of the disease treatment is instigated, the better the response, and determination of the navicular score should be carried out prior to the start of treatment in order to assess the possible outcome.

CHAPTER 6

GENERAL DISCUSSION AND CONCLUSIONS

This present study arose following the hypothesis that vascular occlusion and progressive ischaemia were involved in the pathogenesis of navicular disease (Colles and Hickman, 1977), and was designed to evaluate the radiographic and pathological changes in the navicular bones and surrounding structures in horses in which a clinical diagnosis of navicular disease had been made, and to compare these findings with the results of similar examinations in control horses with no evidence of lameness.

It was concluded from the clinical examination of these horses that no one clinical feature was diagnostic of navicular disease, but that the clinical diagnosis should be based on the presence of a number of clinical signs, and the response to a number of diagnostic aids, in particular, the flexion test and low palmar digital nerve block.

A large number of breeds and almost all work types were represented in the navicular disease group. However, it would appear from this study that the particular type of work performed by a horse does not in itself predispose the animal to navicular disease, but that irregular work and sudden or prolonged periods of rest in an otherwise hard worked horse are predisposing factors. This was first suggested by Turner in 1829 and several workers thereafter (Smith, 1886; Pryer, 1934; Colles, 1982). Smith (1886) considered that intervals of rest in an otherwise hard worked horse resulted in a state of "physiological congestion" of the navicular bone and he concluded that,

If the navicular bone is structurally weak, if it is suffering from the effects of long-continued compression, such physiological congestion is not unlikely to pass into pathological hyperaemia.

Another predisposing factor, suggested by the present study, is the long toe - low heel conformation of the foot. McCunn (1951) considered that such feet were liable to circulatory dysfunction, and Colles et al. (1979) have shown that in a foot with this conformation there was considerable slowing of the rate of blood flow through the foot.

It was concluded from the results of the radiographic examination that no one radiographic feature was diagnostic of navicular disease, since many of the features were also found in the navicular bones of control horses. It is therefore stressed that radiography should not be used to diagnose navicular disease but to confirm a clinical diagnosis. The radiographic features considered important in confirming a diagnosis of navicular disease were, an increase in the number and change in shape and distribution of nutrient foramina in the distal border of the bone, the presence of nutrient foramina in the proximal border of the bone, and radiolucent areas in the body of the bone.

From a study of the development of the blood supply to the navicular bone Colles and Hickman (1977) demonstrated that arteries along the distal border of the bone gradually penetrated the bone, and by the time the horse was 2 or 3 years old, had formed cones of anastomosing vessels, which brought about the development of the so called nutrient foramina visible on radiographs of the navicular bone. Colles and Hickman (1977) also showed that in the navicular bones from navicular disease cases there was an increase in the number of arterioles penetrating the bone from the distal nutrient foramina, which resulted in an alteration of their shape.

Identification and classification of the distal nutrient

foramina in this present study, according to their morphology and anatomical position showed that there were 7 basic types. Cone shaped nutrient foramina in the distal border were considered as normal, and the other 6 types had a significant incidence in the navicular bones of the navicular disease group and were considered abnormal. The changes in shape of the distal nutrient foramina from a normal conical shape were shown, from serial radiographs taken at intervals, to be part of a progressive series of changes.

An increase in the number and change in shape and distribution of the distal nutrient foramina was consistently present on radiographs of the navicular bones of the navicular disease cases, which is considered to be an indication of an increase in the vascularisation of the navicular bone.

An objective method of assessing radiographs of the navicular bone was developed which consisted of a scoring system based on the number, shape and anatomical position of the distal nutrient foramina. Significantly higher scores per navicular bone were recorded in the navicular disease group than in the control group, but a degree of overlap in the scores was found. This overlap was between the scores 10 and 19. It is therefore considered important that a long term investigation should be carried out to determine whether a sound horse whose navicular score lies between 10 and 19 is more likely to develop navicular disease than a sound horse whose navicular score is less than 10.

Pathological examination of the navicular bones and adjacent portion of the deep flexor tendon of control horses and horses with navicular disease showed that pathological changes of different degrees were present in all of the navicular disease cases and in a small

number of the control cases.

These different degrees of pathological change could be fitted into a pattern of increasing severity. The main pathological changes found in the navicular bones were, degeneration of the fibrocartilage, disruption and loss of the subchondral bone plate of the fibrocartilage surface, an overall increase in the vascularisation, and remodelling of the trabeculae in the medulla. The pathological changes found in the deep flexor tendon, which consisted of varying degrees of disruption of the tendon fibres, were considered to be secondary to the changes on the fibrocartilage surface of the navicular bone.

Since a significant positive correlation was found between the degree of gross pathological change present in the navicular bone and deep flexor tendon, and the navicular score as calculated from routine antero-posterior radiographs, it is considered that the navicular scoring system offers a useful indication of the degree of pathological change present.

No evidence of occlusive vascular disease or bone necrosis was found in any of the navicular bones examined in this study.

These results do not support the hypothesis that vascular occlusion and progressive ischaemia are involved in the pathogenesis of navicular disease. It is considered from the results of the pathological examination that hyperaemia rather than ischaemia may be involved in the pathogenesis of this condition. This hyperaemia may be active or passive or a combination of the two.

The results of the treatments used in this study were assessed clinically and radiographically using the navicular scoring system. No significant difference was found between the results of the

different treatments at 3-4, and 6-8 months from the start of treatments, but significantly better results were found with warfarin therapy, 1 year from the start of treatment.

Regardless of the type of treatment to be administered it is considered that the earlier in the course of the disease it is instigated the better the response and that corrective shoeing should be included in the treatment of navicular disease.

It is suggested from the results of this study that the positive effects of warfarin and isoxsuprine hydrochloride in the treatment of navicular disease may not be related to the anti-coagulant or vasodilator effects, but may be associated with their ability to reduce blood viscosity and thus improve blood flow, however this requires further investigation.

The results of this study and of work carried out by Svalastoga (1983) have shown that arterial or active hyperaemia is present in the navicular bones of navicular disease cases, particularly in the subchondral bone plate of the fibrocartilage surface. It is suggested that this may be secondary to mechanical damage or increased functional activity of the subchondral bone plate of the fibrocartilage in response to impact loading or altered mechanical stresses. The predisposing factors of long toe - low heel conformation and/or irregular work could both produce altered mechanical stresses on the navicular bone.

Svalastoga and Smith (1983) have also shown that increased subchondral bone pressure and a lengthening of the pressure drop time were present in the navicular bones from navicular disease cases both of which are consistent with passive hyperaemia (venous congestion) in the navicular bone. In addition a marked slowing of

the circulation time through the foot in navicular disease cases has been demonstrated by angiography (Colles, 1982). It is therefore possible that venous congestion of the navicular bone could develop as part of a general venous congestion of the foot in navicular disease cases.

In either of these circumstances it is considered that the navicular bone pain and so lameness could arise from the disruption of the subchondral bone plate of the fibrocartilage surface as may occur in osteoarthritis in man (Kellgren, 1983) and/or from changes in the intra-medullary vascular pressure (Thurston, 1982).

However it is considered that further investigation is required to establish the origin and nature of the hyperaemia in the navicular bones in navicular disease cases.

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